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PAROXYSMAL HEART BLOCK AND VENTRICULAR STANDSTILL

BY

J. STEWART LAWRENCE AND G. WILSON FORBES

From Haymeads Emergency Hospital

Received August 10, 1943

In auriculo-ventricular block the degree of impairment of conductivity is often found to be inconstant. Even at a time when the rhythm appears clinically to be normal, however, a cardiogram will usually show prolongation of the P–R interval. Only rarely is the conduction normal between the attacks, and the term " paroxysmal heart block " may then appropriately be applied. Reports of 18 such cases have been found, 7 of which had periods of ventricular standstill also; in addition, we found 3 cases of paroxysmal ventricular standstill without heart block (see Starling and Lewis, *infra*).

Hay (1906) described a case in which occasional ventricular beats were dropped, despite regular auricular contractions as shown by the polygraph and unimpaired conduction in the remaining beats; he suggested a depression of ventricular excitability as the cause. His patient was a man of 65 with hypertensive heart disease, and partial heart block had been present in a previous tracing. Gossage (1909) described a similar condition in a woman of 71 with hypertension, and considered it an intermittent form of heart block. In a case recorded by Macintosh and Falconer (1910), a man of 74 had periods of ventricular standstill with Stokes-Adams seizures, during which the auricular rate gradually accelerated, slowing again when the ventricular contractions returned; they were abolished by atropine. Periods of complete A-V block and also of high-grade partial block with a slow irregular ventricular beat were encountered at times. Between attacks, the a–c interval was normal.

Starling (1921) reported the occurrence of periodic ventricular standstill with Stokes-Adams attacks in a man of 51 with a normal-sized heart and a blood pressure of 160/110. Conduction was unimpaired in the intervals. Swallowing used to bring on an attack, but not if atropine had been given during the previous hour. Complete heart block finally supervened. Russell-Wells and Wiltshire (1922) described a male, aged 58, with intermittent complete heart block. The conduction was normal in the intervals. Infective endocarditis was found post-mortem and calcification in the region of the A-V bundle. In the case of Gage and Pardee (1925), a man of 59 with arteriosclerosis, there was intermittent complete block and at other times intermittent ventricular standstill. Conduction was normal in the intervals. Atropine was without effect but adrenaline was followed by ventricular tachycardia, ventricular standstill, and death. Post-mortem, the mitral valve was found to be calcified. Lewis (1925) mentions two cases of intermittent ventricular standstill in patients with normal A-V conduction, in one of which the fibres of the bundle were found to be separated by large venous sinuses. Yater and Williams (1928) described a man of 74 with arteriosclerosis who had periods of normal rhythm alternating with 2 : 1 heart block, complete heart block, and ventricular standstill ; post-mortem, there was calcification in the interventricular septum at the junction of its membranous and muscular parts, and invasion of the bundle of His by fibrous tissue. In the case recorded by Wolferth and McMillan (1928), a woman aged 48, periods of partial and complete block were interspersed with normal rhythm during which conduction was unimpaired. Even during phases of partial block, the P–R interval was normal. Atropine

and amyl nitrite increased the auricular rate and the degree of heart block. Arteriosclerosis was present, and the bundle of His was infiltrated by fibrous tissue and round cells.

The case of Carter and McEachern (1931), a man of 63 with hypertensive heart disease, also had a normal conduction time between attacks of complete heart block—attacks that were unaffected by atropine, exercise, or oxygen. Sachs and Traynor's patient (1933), aged 43, had Stokes-Adams attacks during which complete heart block with a very slow idioventricular rhythm was found; during the intervals the P–R interval was normal; atropine had no effect, but the attacks cleared up following recovery from empyema; there was no evidence of disease of heart or arteries. The patient described by Weiss and Ferris (1934) was a man of 64 with moderate arteriosclerosis and a diverticulum in the œsophagus. Fainting attacks occurred when he swallowed and were found to be due to complete heart block. When a balloon was passed into it and distended, prolongation of the P–R interval was produced, and then complete A-V block, which could be prevented by atropine, or by the injection of procaine into the vagus in the neck so that it was evidently a vagal reflex. Comeau (1937) reported a case of paroxysmal heart block in a woman of 76; the P–R interval was normal between the attacks and these were not abolished by atropine: permanent heart block eventually supervened. A similar case in a man, aged 57, went on to permanent block after two-and-a-half months. Gilchrist (1937) reported two cases of intermittent heart block, both in elderly persons; normal rhythm without delayed conduction was usually followed by ventricular standstill and this by a period of complete A-V block: both patients were benefited by ephedrine. Parkinson, Papp, and Evans (1941) reviewing the subject of Stokes-Adams attacks record the case of a male, aged 43, in whom periods of ventricular standstill started with a delayed auricular beat not followed by a ventricular complex and ended with an ectopic beat 0·1 sec. after P, after which sinus rhythm was resumed without impairment of conduction. Paroxysms lasted 5–20 seconds. There were also occasional periods of complete block. He showed no other evidence of cardiac abnormality and attacks ceased after a month. Of 38 cases of partial heart block with dropped beats recorded by Campbell (1943), 3 had paroxysmal block. One, a woman aged 66 with hypertensive heart disease, had phases of sinus rhythm, dropped beats and complete heart block; the P–R interval was 0·21 sec., which is within normal limits according to Chamberlain and Hay (1939). One in his series had at one time a 2 : 1 heart block with a P–R interval of 0·19 sec. or less, a man of 55 with congestive failure; the third, a woman aged 25, was rather different as the heart block was temporary and due to active rheumatic carditis. [Three others have been added in the paper that follows this—Cases 101, 106, and 115 (Campbell, 1944).]

Observations

Our own case, a retired gardener, aged 71, stated that he had been in good health till four years previously when his ribs were injured in an accident. He noticed some dyspnœa on exertion after this but returned to work. Six months later, however, the dyspnœa was so bad that he had to give up his work and had not returned since. Three years ago he had attacks of nausea, relieved by food; and a few weeks later, attacks that started with a tingling in the legs, passing up to the head, and then severe pain in the head; he would fall forward, dazed but not unconscious. These attacks recurred frequently and two years ago during a severe one he lost his power of speech and had convulsive movements of the limbs. This was the first occasion of his admission to hospital. He was found to have a pulse rate of 35–40 with occasional coupled beats. After an attack of unconsciousness, stated to have lasted 4–5 minutes, his pulse rate rose to 70 per minute. No cardiogram was taken at that time. After discharge from hospital, he continued to have attacks several times a day with occasional remissions lasting a few days. Latterly the attacks have started with pain in the left side followed by palpitation, slow at first but accelerating (probably the auricular contractions). He had to sit down and usually became unconscious for a few seconds; sometimes uncon-

sciousness was more rapid and he fell to the ground. He was readmitted in September, 1942. He was found to be of plethoric type. The pulse was slow and for the most part irregular, varying from 24–56 per minute. The auricular wave could be seen in the neck beating regularly at 80 a minute so that the ventricles on these occasions appeared to be responding to every third beat. Sometimes every beat of the auricles was followed by a ventricular contraction for a minute or so and this would alternate with periods of ventricular asystole during as much as nine consecutive beats of the auricle. During these periods of ventricular asystole, the patient became pale, anxious, and restless, and the auricular waves in the neck gradually became more rapid and forcible. When the ventricles again started to beat, the face immediately flushed, the patient's anxiety and restlessness passed off, and the auricles resumed their previous rate.

The maximum blood pressure was 180/80 mm. The arteries did not appear thickened. There was a systolic murmur, maximal at the apex. No abnormal signs were found in the remainder of the physical examination, apart from absence of the knee jerks and a tremor of senile type in the hands. The urine showed no abnormality. The blood W.R. was negative.

Fluoroscopy showed the heart to be of normal shape and size. On the first occasion the ventricles were responding to every auricular beat, but on a second occasion periods of ventricular asystole were observed and the increasing speed and force of the auricular contractions at these times was confirmed.

Fig. 1, taken on admission, showed an unusual rhythm. There was for the most part, complete dissociation between auricles and ventricles, the auricles beating at 70 a minute, the

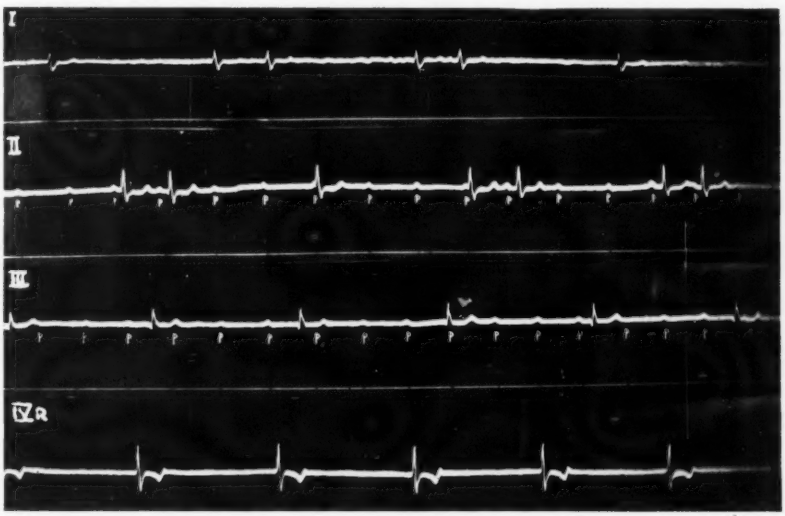

FIG. 1.—On admission. There is complete heart block except when a P wave follows immediately on a ventricular complex when the impulse is conducted normally.

ventricles at 23. At irregular intervals the slow idioventricular rhythm was interrupted by an added ventricular beat which always followed a P wave at an interval of 0·2 sec. These added beats occurred only when the P wave happened to come soon after one of the ventricular complexes. The picture therefore was one of complete heart block, with the ventricles responding to an occasional auricular contraction with a normal conduction time. The ventricular complexes were normal apart from slurring of S I and a biphasic T IV.

After his admission, when no drugs were given, the pulse continued to vary as already described and Stokes-Adams attacks were numerous. Ephedrine was then administered at

4-hourly intervals in increasing doses from 0·5–2 grains. No alteration occurred in the pulse and attacks continued to be numerous. The treatment had to be abandoned in a few days, as it was found to produce retention of urine (the prostate was much enlarged).

Digitalis was then given, the initial dose being half a grain of the powdered leaf, t.i.d., rising to 2·5 grains, t.i.d. Stokes-Adams attacks became less numerous and the pulse, though varying between 20 and 56 a minute, showed less irregularity. Fig. 2 showed that the auricular rate was slower (50) and that, at the time of the tracing, the ventricles were responding to every second beat, the P–R interval being of normal duration (0·18 sec.). Numerous ventricular extrasystoles were present. The P wave was low in all leads but best seen in the right pectoral lead (right arm lead to right arm, chest lead applied at the fourth intercostal space just to the right of the sternum). In the limb leads the QRS complexes had become widened,

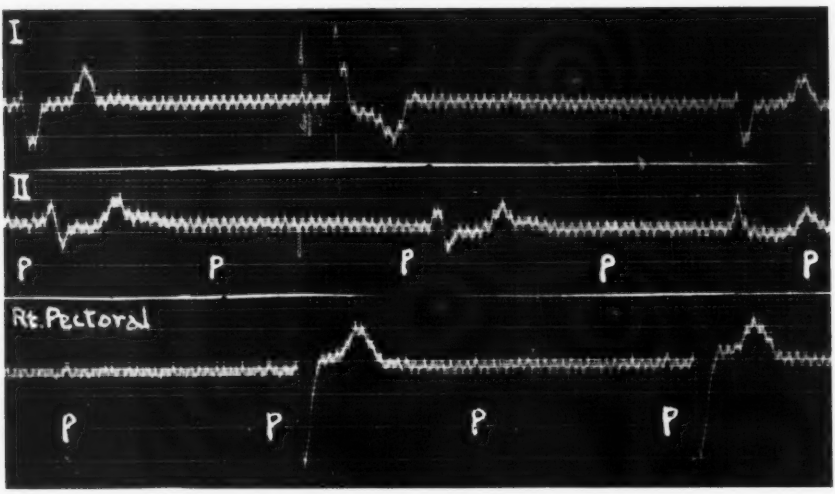

FIG. 2.—The patient is receiving 7·5 grains of digitalis powdered leaf daily. The auricular rate has become slower and the ventricles are responding to alternate auricular beats.

R I and R II diminished, and S I increased. On another occasion, when the patient was still having digitalis, (Fig. 3), he showed a normal sinus rhythm punctuated by periods of ventricular asystole, varying in duration from 6–11 seconds: the auricular rate was 45 and the P–R interval 0·2 sec. During the phases of ventricular asystole the auricular rate steadily increased up to about 60 a minute and then when normal rhythm had returned slowed down to 45 again; the first ventricular systole was not accurately related to the preceding P wave, the P–R interval being 0·3–0·48 sec. and was therefore presumably an idioventricular beat. It was sometimes followed by a missed beat or a ventricular extrasystole before a normal sequence was re-established. As this type of rhythm had been noted before giving digitalis, it was evidently not due to the drug. The inversion of QRST in this and subsequent records could not be accounted for, except possibly by alteration of posture.

Belladonna, 20 minims, t.i.d., resulted in more frequent Stokes-Adams attacks. The auricular rate rose to 90 and, at the time of the cardiogram, the ventricles were responding to alternate stimuli. Such phases were unfortunately very transient and infrequent.

Barium chloride, 1 grain 4-hourly, was without effect. Adrenaline hypodermically merely produced retention of urine.

At this stage it was felt that, as belladonna and ephedrine had not affected the dysrhythmia, it was unlikely to be due to a vagal reflex as in the case reported by Weiss and Ferris (1934). A spasm or partial occlusion of one of the small arteries supplying the bundle of His seemed a

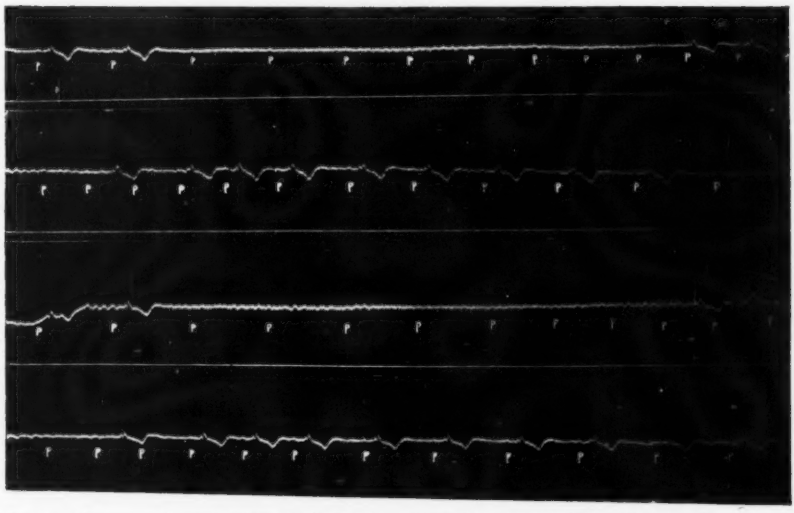

FIG. 3.—Paroxysmal ventricular standstill alternating with normal rhythm (right pectoral lead).

possible cause, so amyl nitrite was next administered. Thirty seconds after inhaling a 2-minim ampoule, the pulse, which before had been beating irregularly at 28, rose to a rate of 70 a minute and became regular, remaining so for the next half hour. In Fig. 4, taken just after the inhalation of amyl nitrite, auricles and ventricles are both beating at 70 a minute, the P–R interval being 0·16–0·2 sec. Just before this tracing, intermittent ventricular standstill had

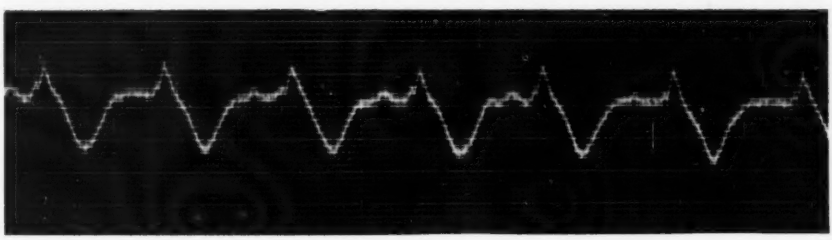

FIG. 4.—Immediately after the inhalation of amyl nitrite. A normal sinus rhythm, rate 70 a minute. The inverted T wave was of similar magnitude before the inhalation (see Fig. 3) (right pectoral lead).

been present as in Fig. 3. Unfortunately glyceryl trinitrini and sodium nitrite were without effect, but by inhalations of amyl nitrite whenever minor attacks began to appear, it was found possible to keep the patient fairly well stabilized. To facilitate this and to avoid the expense of numerous glass capsules, a small wide-mouthed, glass-stoppered bottle was filled loosely with cotton wool and this was soaked with amyl nitrite. The patient kept this by him and inhaled from it whenever the periods of ventricular asystole became troublesome. He soon learned how much to inhale in order to abort the attacks, but no harm resulted if he continued to inhale till the pulse became regular and rapid.

To test further the theory of coronary spasm, the effect of other substances acting on the coronary arteries was studied. Adrenaline dilates the coronary arteries, but also by raising the blood pressure stimulates the vagi through the carotid sinus and aortic nerves (Wright, 1930), so producing an opposing constrictor effect. If the vagus is blocked by atropine, this constrictor effect is avoided and dilatation occurs. Atropine and adrenaline in doses of 0·5 mg. of each were accordingly injected subcutaneously. The pulse rate rose from 30 to 66 in 25

minutes and eventually to 75 where it remained for over two hours. Fig. 5 showed the auricles and ventricles beating normally at 75 a minute, with a P–R interval of 0·2 sec. Ventri-

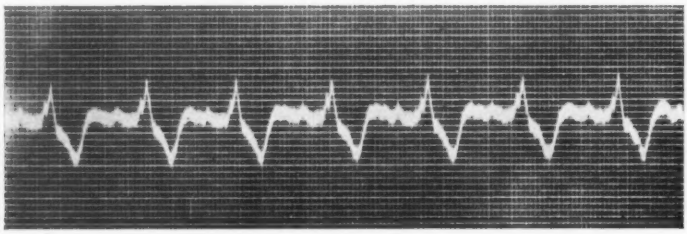

FIG. 5.—After administration of adrenaline and atropine. A normal sinus rhythm is present, rate 75 a minute (right pectoral lead).

cular extrasystoles were numerous at first but these had disappeared after one-and-a-half hours, leaving a pure sinus rhythm.

Both lack of oxygen and excess of carbon dioxide cause dilatation of the coronary arteries. The former, however, does not increase the oxygen consumption of the heart muscle, as the increased coronary flow merely keeps pace with the diminished oxygen saturation (Hilton and Eichholtz, 1925). Thus only excess of carbon dioxide without lack of oxygen will increase the oxygen available to the myocardium. Lack of oxygen was produced in this case by breathing nitrogen through a B.L.B. mask till cyanosis appeared. In Fig. 6, obtained at this

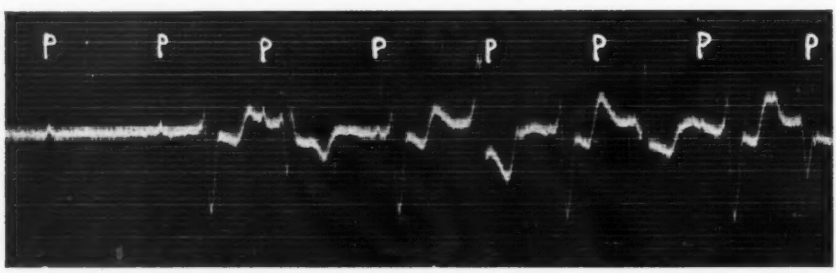

FIG. 6.—Record taken during a period of induced lack of oxygen. There is complete A-V block and ventricular contractions are arising from a number of different foci (right pectoral lead).

time, there is complete A-V block with ventricular complexes arising from a number of different foci.

Carbon dioxide was administered at a concentration of 5 per cent in oxygen. It resulted in the ventricles responding to alternate beats of the auricle, a ventricular extrasystole following each normal beat; the P–R interval was 0·2 sec.

Freedberg, Riseman, and Spiegl (1941) have shown that quinidine reduces the tendency to effort pain in angina pectoris, and prevents the cardiographic changes usually associated with the attack. When this drug was given to our patient in doses of 3 grains 3-hourly, the pulse became, for the most part regular at a rate of 30–32 a minute with occasional more rapid periods (62–70). The cardiogram showed complete heart block, the auricles beating at 75, the ventricles at 33; numerous extrasystoles were present at the time of the record. No Stokes-Adams attacks occurred during the first week of this treatment but during the second week occasional severe attacks occurred. During one of these, the ventricular contractions failed to return and the patient died.

Post-mortem examination showed no gross abnormality in the heart. Small atheromatous plaques were scattered throughout the coronary arteries but these were not sufficient to

obstruct the lumen at any point. No pathological change was evident in the A-V bundle or in any other part of the myocardium.

Discussion

Of the 22 cases reviewed in this paper, 8 had intermittent heart block, 5 had intermittent ventricular standstill, and 9 exhibited at different times both phenomena. In all these cases conduction through the bundle of His was normal between the attacks, even when, as in 4 of the cases of ventricular standstill, only a single beat was missed at a time. These latter cases could be differentiated from partial heart block only by the normal P–R interval in intervening beats. In 4 of the cases, however, transient periods of partial block were noted in addition to periods of complete heart block and ventricular standstill.

The effect of drugs was not uniform. Atropine abolished the attacks temporarily in 4; ephedrine was effective in 2, adrenaline in 1 case. In 1, injection of procaine into the vagus abolished the attacks temporarily. The condition was aggravated by both atropine and amyl nitrite in 1, the only other case in which amyl nitrite appears to have been tried.

Of the patients 13 were males and 5 females, and in 4 the sex was not given. The ages ranged from 25 to 76, the average being 59. Calcification or fibrosis was found in the bundle of His in 4, and large venous sinuses in 1 case. Hypertension or arteriosclerosis was present in 10; in 1, a diverticulum of the œsophagus was shown to be the cause.

The frequency with which intermittent heart block and intermittent ventricular standstill, though both very rare, are associated in the same patient indicates a common cause. An intermittent depression of excitability of the ventricular muscle, as suggested by Hay, explains the periods of ventricular asystole but not the periods of partial or complete block. Moreover, digitalis, which increases the excitability of the heart muscle, does not abolish the periods of ventricular asystole, but seems rather to lengthen them; and quinidine, which depresses the excitability of the heart muscle, does not render them more frequent though possibly more prolonged.

Spasm or partial occlusion of an artery supplying the conducting bundle would account for both the intermittent block and the ventricular standstill, assuming that the bundle fibres were capable of immediate recovery following the short periods of anoxæmia so produced. In favour of this hypothesis, hypertension or arteriosclerosis was observed in half the cases, and all except one were in the latter half of life. Further, in the authors' case, amyl nitrite abolished the attacks in 30 seconds (amyl nitrite takes 30–60 seconds to produce its full dilator effect on the arterioles); atropine and adrenaline together were effective, but not separately; inhalation of carbon dioxide reduced the degree of heart block; and during " complete " heart block, an auricular wave falling immediately after ventricular systole would be conducted through the A-V bundle and give rise to an added ventricular beat. It is in early diastole that the coronary flow reaches its maximum according to Anrep (1926) (for record see Fig. 1). Finally, prolonged ventricular asystole was always followed, after one or two idioventricular contractions, by a phase of normal sinus rhythm. This may be explained as follows. Ischæmia of the A-V bundle due to arterial spasm or partial occlusion blocks the passage of the contraction wave, and ventricular standstill results. This gives rise to a rapidly increasing cerebral and coronary anoxæmia and an accumulation of carbon dioxide in the blood. These three factors all have the effect of dilating the coronary arteries (Hilton and Eichholtz, 1925; Anrep and Segall, 1926), but the coronary anoxæmia still impairs the function of the bundle, and impulses continue to be blocked. Eventually idioventricular contractions appear as in commencing complete heart block. This causes a surge of oxygenated blood through the now widely dilated coronary arteries, thus restoring the function of the bundle. Cerebral anoxæmia and the excess of carbon dioxide are relieved and the tone of the coronary arteries returns so repeating the cycle.

It is unlikely that ischæmia of the A-V bundle is the mechanism responsible for all cases of

paroxysmal heart block and ventricular standstill. A simple inhibitory action of the vagus on the A-V bundle may have been the cause in those cases relieved by atropine, especially the case of Weiss and Ferris in which distension of the œsophageal diverticulum produced an attack, but, as the vagus contains vasoconstrictor fibres to the coronary arteries, ischæmia may have played a part here also.

Summary

A case is described showing periods of intermittent heart block and occasionally a phasic ventricular standstill. Conduction was normal in the intervening stages.

Eighteen reported cases of this type are reviewed.

It is suggested that arterial spasm or partial arterial occlusion is the main factor in the majority of these cases.

It was found possible to control the attacks by inhalations of amyl nitrite.

We wish to express our thanks to Dr. R. A. Hill, Medical Superintendent of Haymeads Emergency Hospital for facilities granted.

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HEART BLOCK WITH ANEURYSM OF THE AORTIC SINUS

BY

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Received August 18, 1943

Disturbances of conduction in the heart may be purely functional, when for example demands are made on the conduction path very early in diastole as seen in blocked and aberrantly conducted auricular extrasystoles (Scherf and Boyd, 1940), or when too many stimuli are presented for conduction within a short time as with the incomplete A-V block that always exists in auricular fibrillation. Conduction disturbances may also be due to vagal inhibition. They may occur transiently after pneumonia and influenza, and are well known in diphtheria; recently Neubauer (1942) found many examples of partial and complete block in 100 cases of diphtheria. Over-digitalization may through poisoning of the A-V node lead to prolongation of conduction time, partial, or complete block. A permanent block can be established through coronary sclerosis, coronary thrombosis, rheumatic heart disease, syphilitic gumma, and more rarely through diphtheria, tuberculosis, and carcinoma affecting the A-V node or bundle directly. Congenital complete heart block is also known, and a few cases were found by Yater (1929), Aitken (1932), Campbell and Suzman (1934), and Currie (1940). The exceptional event of heart block as a result of direct trauma to the chest wall is described by Coffen (1930), Walker (1933), and White (1937).

The following is the report of a case in which complete heart block was associated with an intracardiac aneurysm and aortic stenosis.

A farm labourer, 49 years of age, was admitted to the General Hospital, Northampton, on September 16, 1942. He had been off work since the beginning of July 1942, because of tiredness, dizziness, loss of weight, pains "all over the body," pain and frequency of micturition, and on one or two occasions attacks of hæmaturia. Mainly because of the urinary symptoms he was sent to hospital. Twenty years ago he had diphtheria, but had always enjoyed fairly good health otherwise, except for a regular winter cough. His father and one sister had died of tuberculosis. He was married and had three children aged 1, 7, and 10 years, who were all well; there had been one miscarriage and one stillbirth.

On admission he complained of the above-mentioned symptoms, but his appetite was good and there was no cough, breathlessness, headache, vomiting, abdominal pain, or swelling of the ankles. He was pale and rather thin, but well built. Temp. 99, resp. rate 20, pulse rate 36. There was marked clubbing of the fingers and toes. The cardiac impulse was felt in the fifth intercostal space, one inch outside the mid-clavicular line, and was of a heaving character. A thrill could be felt in the aortic area where there was a loud and rough systolic murmur, which was propagated to the neck and towards the right axilla; a systolic murmur was also audible in the mitral region traceable towards the left axillary line. The blood pressure was 100/75 on the right side, and 120/60 on the left. The pulse was markedly anacrotic and of fair volume in the right arm, while in the left it was of a better volume but not anacrotic. The neck veins were not engorged and showed no abnormal pulsation. The liver was not enlarged; the spleen and kidneys were not palpable. Nothing abnormal could be found in the lungs. There was some slight tenderness in the suprapubic region. The urinary symptoms were proved to be due to subacute cystitis, which cleared up within a fortnight. A radiograph of the chest showed the heart enlarged to the left with rounding of the apex, no broadening of the aortic shadow, and clear lungfields. Fig. 1 taken on September 22 shows a lævocardiogram with regular auricular and regular ventricular activity but continuous variation in the length of the P–R interval. The

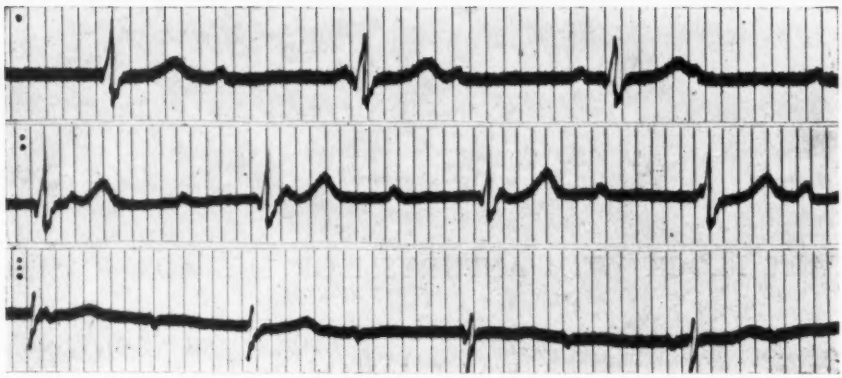

FIG. 1.—Complete heart block and left axis deviation. Auricular rate, 86; ventricular, 40 a minute.

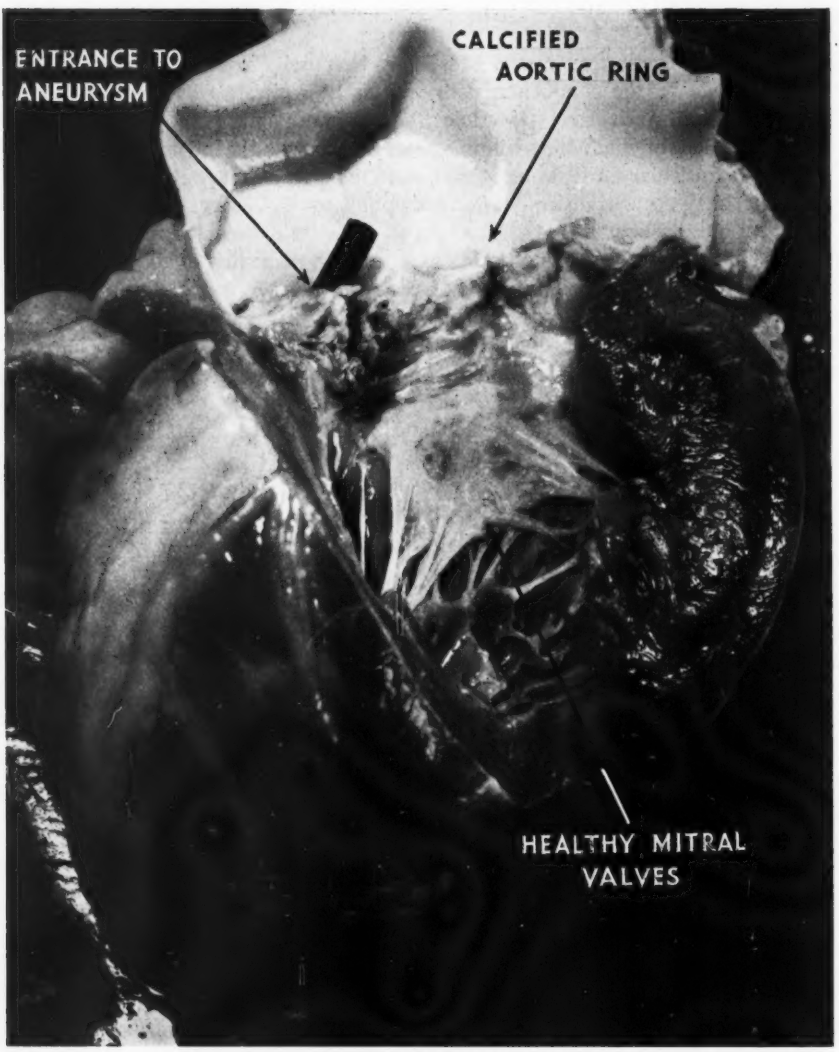

FIG. 2.—View of the heart opened from the front. A rod is placed into the entrance to the aneurysm behind the right posterior aortic valve. The whole aortic ring is severely calcified; the aorta itself and the mitral valve are unaffected.

auricles contract at a rate of 86, the ventricles at a rate of 40 beats a minute. The width and shape of the ventricular complexes (broadened to 0·12 sec. and slightly slurred) cannot be evaluated as indicating a myocardial lesion; the excitation spreads abnormally within the ventricles due to the fact that deep, abnormally located centres have assumed control. There is no displacement of the S–T segment, and the T waves are upright in all leads. The Wasserman reaction was negative.

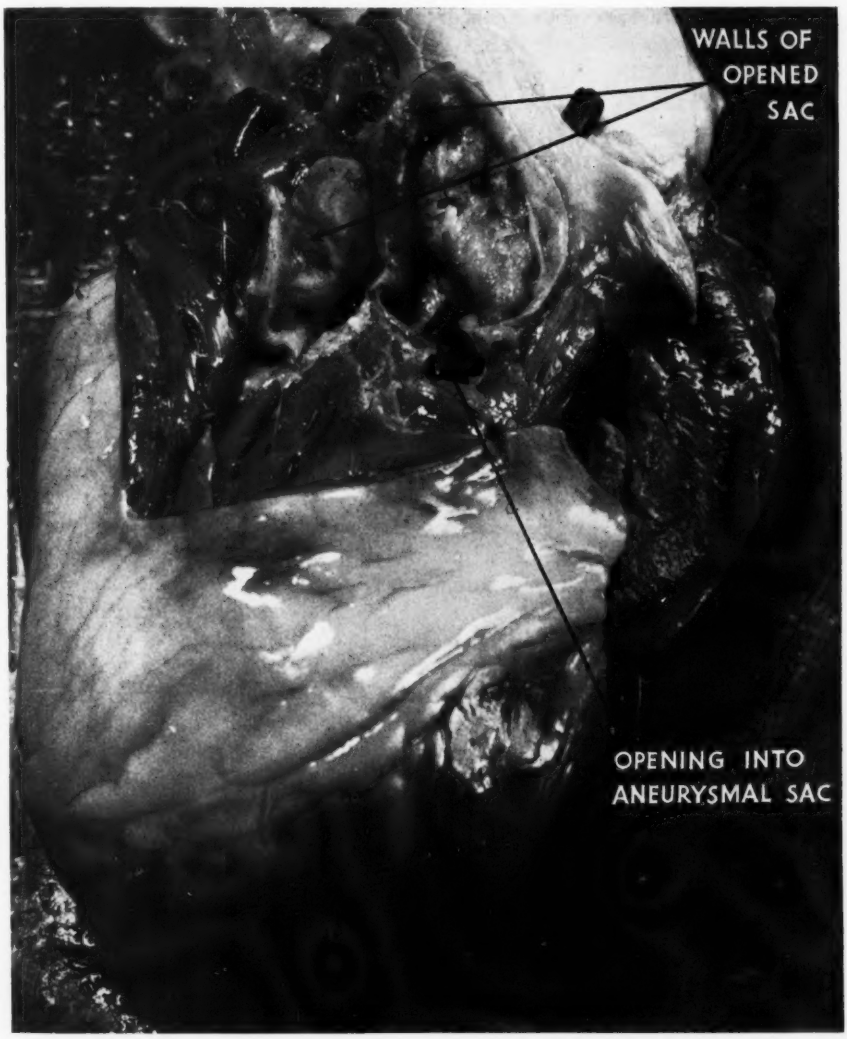

FIG. 3.—View of the right auricle, and of the aneurysm, the walls of which have been cut open and the two halves folded back. The rod appears in the lower part of the aneurysm and shows the communication with the aorta. The two upper arrows point to the two halves of the aneurysm.

Aortic stenosis and complete heart block was diagnosed, and a tentative diagnosis of an aortic aneurysm was considered. The patient was about to be discharged from the hospital on October 1, 1942, when it was noted on the evening of the day before that his temperature was 99·2; the next day it had risen to 100 and remained there until October 5; no cause could be found for the temperature, and the patient felt well and had no complaints. On the morning of October 6 he woke up at 5.30 a.m. feeling clammy and faint, and found it difficult to get

his breath. He was examined ten minutes later. The pulse was of a good volume and regular, but the rate had dropped to 22. He was not cyanosed, but was grey in colour, cold and clammy and very dyspnœic. He improved on coramine and brandy, but three-quarters of an hour later he died suddenly, presumably due to more profound slowing of the ventricular rate with ultimate standstill.

At necropsy the brain was not examined. The lungs were voluminous, but not congested, they appeared to be normal, apart from a healed apical lesion on the left side. The pleural cavities were normal. The pericardium contained some clear fluid. The heart was hypertrophied, mainly in the left ventricle, and the right auricle was dilated. The tricuspid, mitral, and pulmonary valves were normal. A saccular aneurysm of about 1 1/2 inch diameter was found projecting into the right auricle, lying on the lower part of the interauricular septum and communicating with the aorta through a 1/4 inch diameter passage immediately above the right posterior aortic valve. There was severe calcification of the aortic valve ring which was continuous with the wall of the aneurysmal sac (Fig. 2 and 3). The aneurysm was not ruptured. The aorta and blood vessels, including the coronary arteries, showed no atheroma. The liver and spleen were slightly enlarged and showed early venous congestion. The gastro-intestinal tract and the pancreas were normal.

Discussion

The ascending part of the aorta is normally a little enlarged just above its origin; this enlargement consists of three small pouches, known as the sinuses of Valsalva, opposite to which are attached the three semilunar valves. Aneurysm may develop here and involve one, two, or all three of these sinuses of the aorta. They rarely attain a size to produce physical signs, and before symptoms appear can lead to sudden death, which may be of medico-legal interest. Death occurs through perforation, either into the pericardium or more rarely into the superior vena cava, pulmonary artery, or right or left auricle. Syphilitic aortitis is usually the cause but a few cases were found with ordinary atheromatous changes. The aortic valves are as a rule affected. Whilst I am not acquainted with a publication of a case similar to the above described, I found a reference to the aneurysm of the sinus of the aorta by Matthes (1928) who states that it is a very rare occurrence, that it affects usually the right sinus, and that it may develop into the right ventricle, more rarely into the right auricle, or outwards and upwards. He further mentions that these aneurysm can be diagnosed only if they burst, and thus lead to a conspicuous alteration of the whole clinical picture, e.g. the sudden occurrence of a very loud murmur which extends over both heart phases, and was not present previously. In any case one can only make the diagnosis if one knows that the aortic valves are diseased.

Reference should also be made to the case of Micks (1940) who found aneurysms of all three sinuses of Valsava; the patient developed complete heart block before his death.

In trying to find the cause of the heart block it appears that four possibilities have to be considered.

1. The absence of any fainting attacks may suggest that the block was congenital. The patient was carefully interrogated as to any previous loss of consciousness, etc., but denied firmly any such occurrence. On the other hand, he volunteered the information that his fingers were clubbed as long as he could remember. However, the postulates of Yater (1929), concerning congenital heart block, are not completely fulfilled, and in view of the other findings this possibility is not very likely.

2. He had diphtheria twenty years ago. There is as yet no conclusive evidence as to the after-effects of diphtheria on the heart. In 100 cases which had suffered from diphtheria 5 to 10 years previously Jones and White (1927) did not find any signs of heart disease. Hoskins (1926), on the other hand, found persistent cardiographic changes quite common after diph-

theria, and Butler and Levine (1929) found a history of diphtheria in 10 out of 20 patients with heart block of obscure origin, and believe that diphtheria is of ætiological significance in many such cases.

3. The aortic valve ring was severely calcified, causing aortic stenosis. Auricular-ventricular or bundle branch block is an occasional complication of aortic stenosis; this may be due to extension of the calcifying process from the aortic ring into the interventricular septum with implication of the bundle of His. Unfortunately a histological examination of the bundle was not made in this case.

4. The aneurysmal sac was situated close to the auricular part of the Aschoff-Tawara node, and it is most likely that the pressure of the aneurysm on the node caused the block. On this assumption the post-mortem report as to the cause of death was as follows : Aneurysmal sac of aorta, pressing on bundle of His, secondary to ulcerative changes in the aortic valve ring.

Summary

A case is reported with an aneurysm of a sinus of Valsalva and complete heart block. The possible causes of the block are discussed, and the conclusion is drawn that the block was due to pressure of the aneurysm on the conducting path.

My thanks are due to Dr. W. M. Robson for permission to report the case, and to Dr. Margaret Masson for the photographs (Fig. 2 and 3) and most helpful co-operation.

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STOKES-ADAMS ATTACKS IN A CHILD

BY

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It is recognized that heart block may occur in several acute infections, but apart from diphtheria and acute rheumatism it is rare and even in the latter it is uncommon. Often the outcome of this complication in children is fatal, but in the following case, a boy aged 12, with heart block and pericarditis, recovery took place.

For three months before admission to hospital he had felt tired and had to rest on returning from school. He had been restless for the past few nights and eventually had to remain in bed for three days. During this time he had throbbing of his heart and headache. For two days he had a troublesome unproductive cough, and on the day before admission he had two attacks of unconsciousness. On coming round he perspired and vomited. He had one attack of unconsciousness the day before coming to hospital and on admission he complained of giddiness, shortness of breath, and persistent coughing. Apart from measles there was no history of any previous illness.

On admission the patient was very ill. He presented grey cyanosis and was distressed by severe dyspnœa. The temperature was normal and the pulse was small and 160 a minute. The blood pressure was 80/40. The respiration rate was 44. There was prominent pulsation and distension of the cervical veins. The apex beat was diffuse and was just outside the mid-clavicular line. The heart sounds were distant and the second sound was split and accentuated in the pulmonary area. There were no murmurs. Numerous rales were heard over the chest. No other abnormal sign presented on further examination; there was no distension of

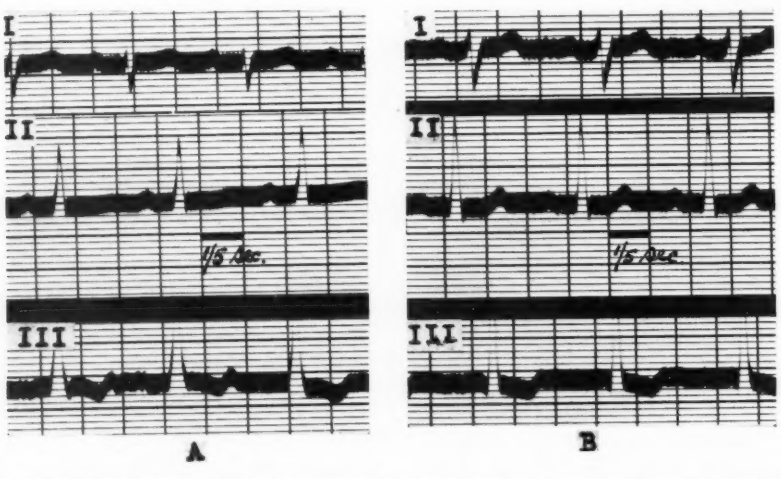

FIG. 1.—Electrocardiograms. (A), taken two weeks after admission, shows flat T waves in lead II and inverted T waves in lead III; (B), taken six weeks later, shows an upright T wave in lead II.

liver and no ascites or œdema. The diagnosis of acute rheumatic pericarditis was considered likely. The patient's general condition deteriorated and he was given oxygen by nasal catheter. Next day the pulse rate dropped to 48. The diastolic blood pressure rose to 70 but the systolic was difficult to record. On the morning after admission, I witnessed two Stokes-Adams attacks. They were preceded by restlessness, an anxious expression, complete irregularity of the pulse, and quick and deep breathing. The pulse, the heart sounds, and later the respiration, ceased. Pallor gave way to cyanosis. There was no twitching. No pulsation of the vessels of the neck was observed during the cardiac standstill. Unconsciousness lasted for ten seconds. After coming round the patient became very flushed and perspired profusely. He was given ephedrine and digitalis and the pulse remained at 80 to 90.

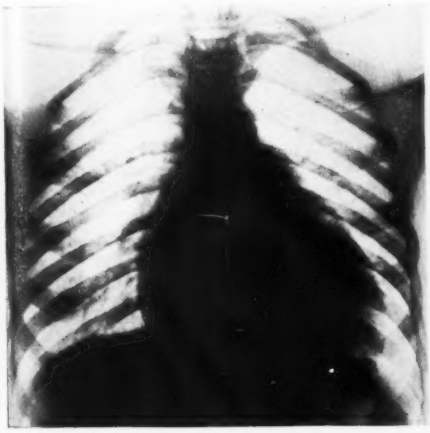

FIG. 2.—X-ray of chest one day after admission to hospital.

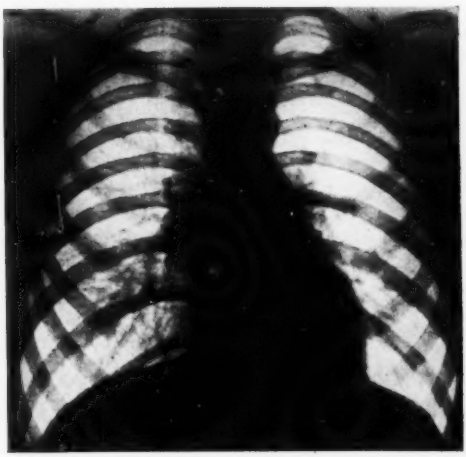

FIG. 3.—X-ray of chest eight weeks after admission to hospital.

During the next three days he had further attacks, but these became less frequent and the patient improved. A pericardial rub appeared on the third day and lasted for three days. At no time did he have any pyrexia or œdema and his later recovery was uneventful. His blood pressure rose to 110/70 and his pulse remained regular at 90. No electrocardiogram was taken during the period of irregularity and later it showed changes due to pericardial effusion with a normal P–R interval (Fig. 1).

Radiography also confirmed the pericardial effusion (Fig. 2 and 3).

DISCUSSION

It is known that primary carditis in childhood is often the first and only manifestation of rheumatic infection. Though Cheadle (1889) and later Moore (1909) stated that carditis was the central factor in rheumatic infection in childhood the diagnosis was often impossible owing to the absence of specific symptoms. In my case the pericarditis was probably rheumatic in origin and its presence was confirmed by the pericardial rub and later cardiographic and cardioscopic findings.

According to Lewis (1925) the cause of clinical heart block is firstly a lesion of the conducting tract, secondly vagal stimulation, or thirdly the result of poisoning. The first cause was operative in my case and the evidence points to heart block caused by rheumatic infection. It is obvious that this infection must have involved the myocardium and the pericardium but there was no evidence of endocardial involvement. The recovery of the patient precluded any pathological examination of his tissues, but certain conclusions can be drawn from the

clinical examination. In the cases that have come to necropsy there have usually been found inflammatory infiltration with rheumatic giant cells and fibrotic changes. In some cases there has only been exudation in the collagenous tissues of the membranaceous septum. Gross and Fried (1935) in 110 necropsies on rheumatic hearts frequently found inflammatory changes in the collagenous extension of the fibrous septum which abuts on the bundle tissue. In many of their cases a portion of the conducting system was entirely surrounded by relatively rigid collagenous tissue and they considered that this, when expanded by exudation, might compress the conducting fibres. It seems reasonable to suppose that in my case as there was no permanent lesion of the conducting system, the pathological change was in the nature of either a minor inflammatory condition of the bundle tissue itself or of such exudation which pressed on the bundle.

Summary

A case of insidious rheumatic pericardial effusion with Stokes-Adams attacks is described. A boy of 12 sought medical help after three months of ill health and only when disturbance of the conducting system had set in. Either minor inflammation of the bundle tissue itself or its compression by inflammatory vascularization of the collagenous mass in the septum membranaceum, would explain the symptoms.

I should like to thank Dr. Fiddes, Medical Superintendent, E.M.S. Hospital, Billericay, Essex, for facilities and Dr. William Evans for helpful advice.

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COMPLETE HEART BLOCK

BY

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Received October 21, 1943

This paper discusses 64 cases of heart block, mostly complete, seen during the same period as 29 cases with dropped beats and 140 cases with latent heart block (Campbell, 1943, *b* and *c*).

Of the patients attending the Cardiographic Dept., Guy's Hospital, during 12 years, 0·6 per cent had complete, 0·5 per cent partial, and 2·2 per cent latent heart block. Comeau (1937) found 0·55 per cent of cardiovascular cases had complete block; and Paul White (1937) found 0·8 per cent had complete, 2·6 per cent partial, and 3·0 per cent latent. It is curious that with such close agreement between the other figures he should have found so many more with partial block.

An analysis of the age and sex of the cases and of their actiology was similar to that found in other series, so this and the clinical features will be dealt with shortly. Three aspects will be dealt with rather more fully—the prognosis, the incidence and significance of Stokes-Adams attacks, and the extent to which complete block remains persistent or varies to lower grades of partial or latent block. This leads to a more complete classification of the subdivisions of heart block.

I had the impression that, apart from the cases in hospital with frequent changes of rhythm and Stokes-Adams attacks, complete heart block was as a rule persistent once it had been found. But half my cases had more than one grade of heart block graphically recorded; and more than 60 per cent were observed clinically to have such changes. Sometimes these were frequent and one did not know with what rhythm the patient would be seen next: sometimes, after a long period of complete block, partial or latent block was seen for no apparent reason.

Aetiology

Sex incidence. There were 51 men and 13 women, a preponderance of 4 : 1. If the 3 rheumatic cases (all women) were excluded the proportion rose to 5 : 1. Graybiel and White (1936) found the proportion 2 : 1; and all observers are agreed that it is much more common in men.

This is another contrast between complete and lower grades of heart block where the difference of sex incidence is absent or much less marked. In my 38 cases with dropped beats, 19 were men and 19 were women, and this applied as much to the elderly myocardial group as to the younger patients with rheumatic and other infectious diseases. In the 31 cases with latent block with a P–R interval over 0·26 sec. or more there was a preponderance of males, but it was entirely in the elderly group. There were 19 younger patients (average age 24) with rheumatic and other diseases, and here there were 11 males and 8 females: there were 12 older patients with myocardial disease (average age 56), and here there were 10 men and 2 women—the same proportion as with complete block.

Age incidence. The age, reckoned from when they were first seen (and generally this seemed to be near the time of onset), is shown below.

Age	Under 40	40 to 49	50 to 59	60 to 69	70 and over
Number of cases	5	5	17	29	8

Most patients (45 per cent) were between 60 and 69, and 84 per cent were over 50. In the small number under 40, the prognosis seemed to be better, but otherwise the age did not seem to be important. The better prognosis at the younger age would, of course, be made much more striking had congenital cases been included. A more detailed analysis is given in Table I. Originally those with and those without latent block were listed separately, but as there was no significant difference they have been combined.

TABLE I

AGE OF PATIENTS WITH HEART BLOCK (AGE WHEN FIRST SEEN)

	Under 40 years	40 to 44	45 to 49	50 to 54	55 to 59	60 to 64	65 to 69	70 to 74	75 and over	Total
Patients known to have died ..	1	2	2	3	4	8	7	5	2	34
Patients still alive when followed ..	4	—	1	1	3	4	3	—	—	16
Patients not traced	—	—	—	4	2	3	4	1	—	14
Total	5	2	3	8	9	15	14	6	2	64

Ætiology. Myocardial disease without any evidence that it was rheumatic or syphilitic was much the most common cause—86 per cent of all cases, or 76 per cent if congenital cases were included. The congenital cases, which have been excluded otherwise from this paper as they have already been reported (Campbell, 1943*a*), were the second commonest group, forming 13 per cent of the total, while the rheumatic and syphilitic groups together formed only just over 11 per cent.

The figures found in the series collected by Graybiel and White (1936) were so similar that I have included them for comparison in Table II. The only significant differences were that they have included a few cases in a diphtheritic group, of which I have not found any examples, and had left more cases as due to combined causes (where I had more dogmatically placed the case in one group or the other according to the factor that seemed most important), and that I had more congenital cases. The average ages of the different ætiological groups have also been added.

TABLE II

AETIOLOGY OF COMPLETE HEART BLOCK

Aetiology	Number of cases		Average age	
	This series	Graybiel and White	This series	Graybiel and Whit
Rheumatic	3	3	34	41
Syphilitic	5	3	42	43
Myocardial	56*	47	64	63
Congenital	10	4	4 †	6
Diphtheritic	0	4	—	35
Combined causes	0	11	—	—
Total	74	72	—	—

* High blood pressure, 10; coronary atheroma, 17; cardiac failure, 4; and cardiac enlargement generally with atherosclerosis without the previous features, 25.

† The present age of the seven known to be alive is 30 years.

High blood pressure has not been taken as a separate main division and my evidence for calling a case myocardial must be given. Of the 56, 10 had high blood pressure (over 160/100). In several others the systolic pressure was high, but as this seems to rise in many—but by no

means all—cases to compensate for the long diastole, this was not counted until the diastolic pressure was also raised; 17 were included as having coronary arterial disease and the evidence for this was a history of angina pectoris or cardiac infarction; and 6 had congestive heart failure without high blood pressure or evidence of coronary disease.

The remainder had enlargement of the heart without these other findings: it is surprising that cardiac enlargement was the only other decisive evidence of heart disease in 23 of the 56, though many had thickened radial or retinal arteries and still more had signs of atherosclerosis of the aorta on radioscopy. It is surprising and significant that so many patients with complete heart block seem relatively well and are able to lead quiet lives, almost normal for their age, provided they do not suffer from Stokes-Adams attacks. One common type is the elderly man with grey hair and thickening arteries who has led a strenuous and sometimes intellectual life, often with hardly a day's illness.

The rheumatic cases were few: one with mitral disease had varying grades of heart block that were occasionally complete (Case 107, p. 80), one had temporary block during active rheumatic carditis (Case 113, p. 83), and one was called rheumatic on a history of rather doubtful rheumatism in childhood (Case 140, appendix). Their average age was 34 years. Perhaps a fourth should have been included, but on his age of 68, on the late onset of any symptoms before his congestive failure, and on the signs being predominantly those of aortic stenosis, he was included as atherosclerotic though he had also some aortic incompetence and a history of rheumatic fever 38 years before (Case 150). The two other cases with aortic stenosis are referred to on p. 76.

Case 150. A man, aged 68, had rheumatic fever when he was 30. He got on well, though with increasing dyspnœa as he got older, till at 68 he was admitted to hospital with congestive heart failure. He was found to have aortic stenosis and incompetence, C.H.B., B.B.Bl., and auricular fibrillation (Fig. 1); his B.P. was 190/60; his W.R., negative. He improved for a time but died five months

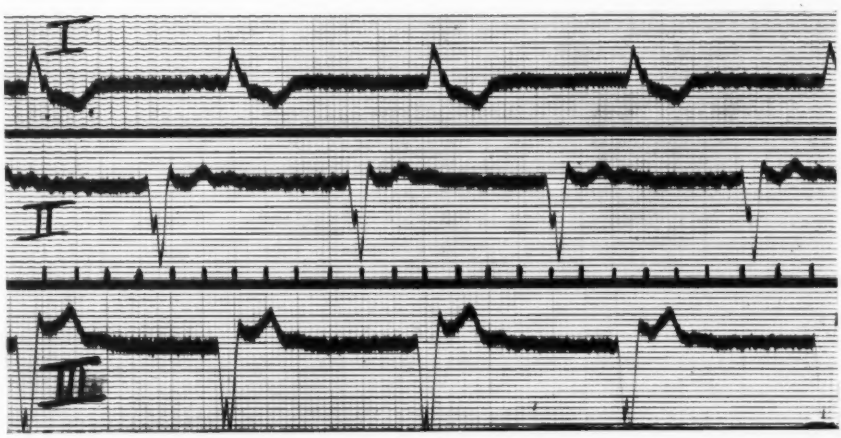

FIG. 1.—A faster rate than usual, in an old man with atherosclerosis are aortic stenosis. His rate averaged 48 and was 47 a minute in this record, when he had also, as usual, bundle branch block and auricular fibrillation (Case 150). In this and most subsequent figures the inked marks indicate 1/5th sec.

later, after developing coupled beats of an irregular type, probably as the result of treatment with digitalis (Fig. 2). Post-mortem, there was left ventricular hypertrophy with extreme calcification of the coronary arteries. There was calcified thickening of the aortic cusps and old fibrosis of the mitral valve.

The striking thing about the syphilitic cases was that none had the usual signs of aneurysm or gross aortic incompetence. One only had a slight aortic leak and he gave no history of syphilis and had a negative Wassermann reaction (W.R.) (Case 123, appendix). One was

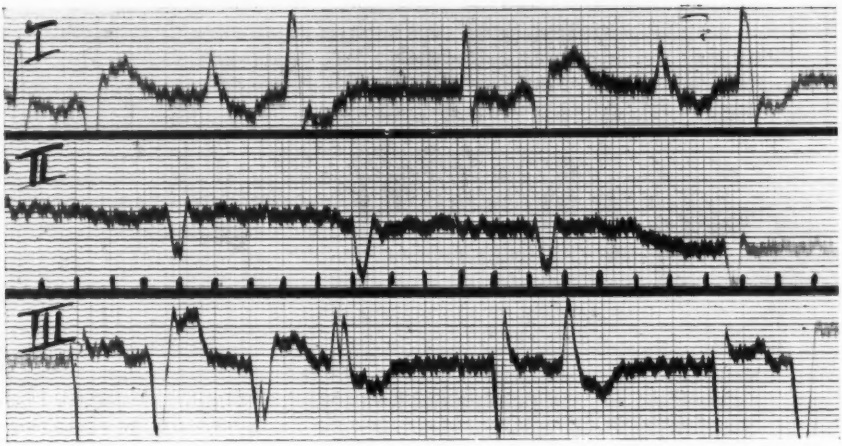

FIG. 2.—Bigeminy of an irregular type from the same patient as Fig. 1. It was taken some months later, shortly before his death, and may have been partially due to digitalization. In lead II there is C.H.B. with a still faster rate of 54 a minute; in leads I and III, bigeminal extrasystoles of different types and also changes in the bundle conduction.

included on a positive W.R. and signs of neurological syphilis though his blood pressure was 250/130 (Case 101, p. 83). One was included on a positive W.R. only (Case 138). One may have had congenital syphilis as her father had died with general paralysis of the insane, but there were no other signs of this and her W.R. was negative (Case 159, appendix). The last, aged 34, was included on the sole evidence of a weakly positive W.R. (Case 108, p. 81). The average age of these five patients was 42. Even in these cases it will be agreed that it was not really certain that the heart block was caused by syphilitic disease.

There were three others where this diagnosis had to be considered. One had a positive W.R. but also had a blood pressure of 210/130 and albuminuria, and post-mortem his heart weighed 900 g. and there was no evidence of syphilitic disease (Case 120, p. 81). Two others had angina and arteriosclerosis and were classified as this, although one had a positive W.R. (Case 104) and the other a history of syphilis with a negative W.R. (Case 119), because there was no evidence on radioscopy or otherwise of syphilitic disease.

A possible explanation of the absence of typical syphilitic cases is that these develop in the quaternary stage of the disease, and that heart block, when it is syphilitic, is more likely to arise in the tertiary stage as the result of a gumma affecting the A-V bundle.

There is unusual doubt about how many of these cases should be included, but if every doubtful case were included as rheumatic or syphilitic the incidence would only be increased to 5 and 10 per cent respectively.

STOKES-ADAMS ATTACKS

Stokes-Adams attacks are found in about half the patients who attend hospital with complete heart block, and probably this gives a true picture of their incidence. When present they form the most striking part of the clinical picture. They may be few and far between, or very frequent, or frequent at first and rare or absent later.

The patient who has not had an attack when first seen need not, however, be unduly alarmed about them. Probably he will never have an attack, and after six months freedom their onset becomes much less likely (see p. 74). His outlook must therefore be judged on the presence of congestive failure, angina pectoris, etc. Except for the statistical evidence

that follows I have no help to give for the prognosis of those who have these most alarming and unpredictable attacks.

It is not easy to decide exactly what should be included as Stokes-Adams attacks. Should the minor attacks in which consciousness is not lost be included, as these are obviously due to the same mechanism but of shorter duration? Should similar attacks in patients without heart block be included? In order to give a precise meaning to the term, I think they should not and would accept the definition of Parkinson, Papp, and Evans (1941): attacks of loss of consciousness due to ventricular standstill, ventricular tachycardia, ventricular fibrillation, or a combination of these, in a patient with heart block. It is most important to add that the heart block may be established or paroxysmal, and as the paroxysmal heart block may only occur for a short time after the attack, the diagnosis must sometimes be made on clinical grounds only without a proof of the heart block when no precise record of the behaviour of the heart during and after the paroxysm is available. The attacks are of two main forms, syncopal or epileptiform, this probably depending on the duration of the arrest of the heart's action.

It is sometimes thought that the onset must be absolutely sudden, as it obviously is in most patients who fall so that they hurt themselves. But in some patients a shorter arrest of the heart's action—too short to produce a true attack—would produce a feeling of faintness and other symptoms; and in such patients these symptoms will often be present as an aura to the fully developed attack. By chance the first two patients quoted by Parkinson *et al.* illustrate these two possibilities, and we have many examples.

Case 4 of Parkinson *et al.* proves, if further proof were needed, that the minor attacks are of the same nature. After various symptoms "he expected to faint but only turned pale and breathed rapidly. In some of them the pulse stopped and the ventricles ceased beating for 5 seconds or more." . . . " In almost as many attacks consciousness was lost and the ventricular standstill lasted 10–20 seconds, the patient becoming unconscious towards the middle of this period."

This paper of Parkinson, Papp, and Evans has dealt so fully with the actual behaviour of the heart during the attacks that I have nothing to add except a description of one case of Dr. Suzman's (see appendix). But in most cases cardiographic records are not available and the diagnosis must be made on the story. Generally there is not much difficulty in patients with the classical abrupt onset who fall and hurt themselves, unless the heart block is missed because it is paroxysmal. But the preliminary aura may lead to confusion with fainting attacks, vaso-vagal attacks, or even hysteria, though the sensible matter-of-fact patient generally prevents this.

The aura may be very variable and a few random examples are given in the patients' own words. " Can't breathe, pins and needles all over the body, then remember nothing " (Case 107). " Feel rushing in the ears, then dazed for a few seconds, then trembling, then all right again—would fall down if I had nothing to hold, and can sometimes remember the actual fall " (Case 108). " Go blind, feel queer and giddy *and as though my heart has stopped*, and then fall down unconscious " (Case 130). [I have put some of these words in italics because I think that many patients are remarkably aware of visceral happenings, and that some doctors are not ready to pay enough attention to the patient's description of his sensations, on which most of our diagnosis rests.] " Severe precordial pain passing up the left side to the lower angle of the jaw, then fall unconscious " (Case 142).

Case 123 (appendix) gave a most curious story and no diagnosis was made for years ; even when he developed heart block I did not feel sure of the nature of his attacks which were probably associated with paroxysmal heart block at first, until I had become more familiar with this condition.

It is sometimes said that biting the tongue and incontinence of urine or fæces are diagnostic of epilepsy and exclude Stokes-Adams attack. This is not so, though these events are rare

in Stokes-Adams attacks and common in epilepsy. In Case 7 of Parkinson *et al.* there was incontinence of urine and in Case 6 incontinence of fæces. The occurrence of tongue biting and incontinence of urine in my Case 142 can not therefore establish the original diagnosis of epilepsy that was made in this patient. Probably the duration of the ventricular standstill is the decisive factor.

Minor attacks. Between true Stokes-Adams attacks and the attacks of faintness and giddiness that are not included as such, there are minor attacks which have the same relationship to true attacks as petit mal to grand mal. Case 116 provided a good example. In one major attack he " felt it coming, but had no time to get to the railings and fell and cut his head." In a minor attack he " felt a little funny on his way home from work, had a momentary blank, but the paper did not fall from under his arm."

It is not easy to see why there should be such a variety of symptomatology. In epilepsy the varying auras can be explained by different spreads of the nervous impulses through the brain, but in Stokes-Adams attack the heart is at fault and one might guess the central effect in the brain would always start and spread in the same way. But patients with Stokes-Adams attacks have generally atherosclerosis, and probably different degrees of atheroma in different cerebral vessels influence the effect that ventricular standstill has on the functions of the brain.

Presenting symptoms. There was a striking difference in the presenting symptoms of those with and of those without Stokes-Adams attacks. Most of the latter were first seen for dyspnœa or attacks of faintness or giddiness.

Of the former, 21 (63 per cent) were first seen because of the Stokes-Adams attacks and they had no serious cardiac symptoms before this. It does not, of course, follow that the attack was actually at or near the onset of complete heart block (C.H.B.) but it seems likely that this was so in many cases; 2 others had coronary thrombosis which was quickly followed by C.H.B. and Stokes-Adams attacks, though in one of these the slow pulse was noted the day before the first attack.

There were, on the other hand, 8 cases where the heart block was known to have been present for a reasonable period before there was a true Stokes-Adams attack: 6 of these had attacks of faintness or dizziness as the first presenting symptom, 1, 1, 3, 4, 8, and 24 months before respectively (" last two years have had several giddy turns, too many to count; last week fell and cut my head ", Case 116); one other complained of dyspnœa only and 6 months later had his first attack, and the eighth came to hospital for gangrene of his leg and had his first attack 7 weeks later.

The remaining 2 cases were under observation for dyspnœa and angina before they developed C.H.B. The former was readmitted to hospital with congestive failure and C.H.B. and the first attacks which soon proved fatal followed 5 months later (Case 120). The latter was found to have C.H.B. five weeks after her first Stokes-Adams attack and as this was the first time she had been seen since the attack it probably indicated the origin of the C.H.B. (Case 118, p. 81).

Thus the Stokes-Adams attacks were the first significant symptom of C.H.B. in 72 per cent; and in 27 per cent the C.H.B. was known to have been present, or other symptoms made this likely, for an average period of 6 months before there was a Stokes-Adams attack.

The exact onset of complete heart block is often unmarked by any specific symptom where the patient does not suffer from a Stokes-Adams attack. In certain patients, however, it can be recognized, and I have three histories where there was some confirmatory evidence. " Sudden thumping in the ears, nearly lost control, but was not unconscious "; his doctor found the slow pulse the same afternoon (Case 134). " Started with onset of giddiness, nearly fell down, noticed breathlessness the same day " (Case 162). " Attacks of flushing, giddiness, and discomfort, without loss of consciousness, starting when my heart became

slow." This should be accepted, as after six years of established complete heart block she came to hospital one afternoon saying that her heart had gone back to its normal rate and this was found to be the case (P–R, 0·23 sec.), but it did not persist: high blood pressure may have made her unusually aware of her heart rate (Case 125). Of the 31 who had not Stokes-Adams attacks, dyspnœa (10 cases) or faint or giddy attacks (12 cases) were almost equally common presenting symptoms, and between them accounted for 70 per cent of the cases. Of the others, three had congestive failure, one angina pectoris, and one coronary thrombosis. In the remaining four the presenting symptoms were varied and often non-cardiac and the finding of complete heart block was a surprise.

Other Features of the Clinical Picture

Heart rate. This was carefully analysed in 30 patients in whom I had frequent records over a reasonably long period—sometimes for many years. Taking an average figure for each case, the results are shown in Table III.

TABLE III

Heart Rate in Complete Heart Block

Heart rate	Under 24	24 to 27	28 to 31	32 to 35	36 to 39	40 to 43	44 and above
Number of cases	1	3	6	4	9	5	2

In the great majority the average rate was between 28 and 40 with a grand average of 34·6 a minute. This is slower than the rate of 38–40, given by Graybiel and White (1936) and I think more in accord with general experience. Of course there was variation in an individual case from time to time, and typical figures were 24–30, 28–36, 32–40, and 36–42; in the two patients in whom there were the largest number of observations over many years, the range was 32–44 with two single readings of 45 and 54, and 28–40 with an average figure of 34·8.

The specially fast and slow rates were considered for all the patients. Two had average rates of 24 (Cases 144 and 146) and both were specially liable to feeling faint and giddy apart from their true Stokes-Adams attacks. One, who died three days later in a Stokes-Adams

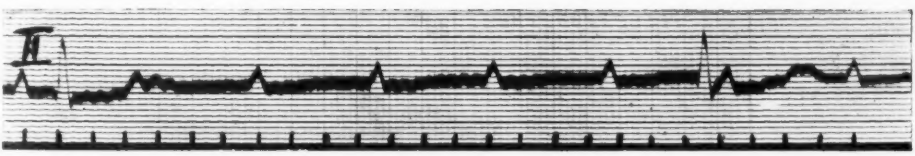

Fig. 3.—An unusually slow rate in complete heart block. His rate, clinically, was often 18–20 and here in lead I it was as slow as 15·8 a minute (Case 155).

attack, had a rate of 18–20 and sometimes a cardiographic rate of 17 (Fig. 3); curiously enough he did not complain specially of any symptoms while this record was taken (Case 155).

In several patients a slower rate than their usual one was often associated with regular bigeminal ventricular extrasystoles (Fig. 4) and was perhaps their cause, though it is not clear how they arise in complete heart block. For example, one whose rate was generally 32–44 had a rate of 56 with alternating extrasystoles, i.e. a true rate of 28; and another whose usual rate was 28–40 had alternating extrasystoles when the rate without these would have been 22.

Rates over 44 were rather more common but there were special features about the four patients in whom these were found regularly at rest. A girl, aged 18, is reported in the appendix (Case 159). In a young woman, whose heart block was temporary and due to acute rheumatic carditis, the rate was 62 (auricle 115) (Case 113, p. 83); this rapid rate with complete heart block is well known in diphtheria.

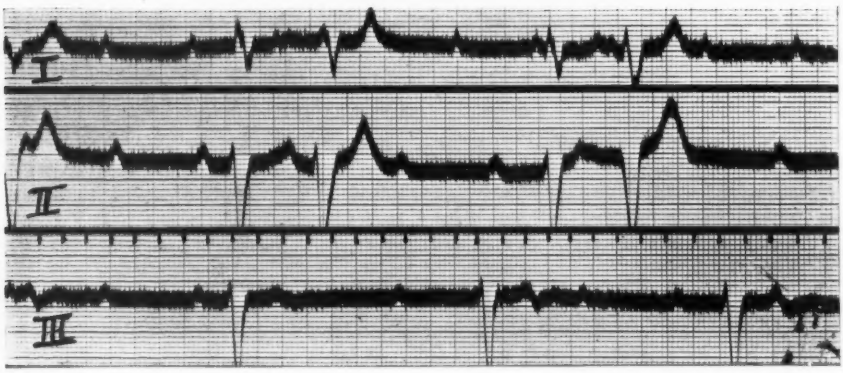

FIG. 4.—Regular bigeminal extrasystoles with complete heart block. In lead III the ventricular rate is a little faster and there are no extrasystoles (Case 132).

In two cases with aortic stenosis and incompetence, the rate was between 36 and 50 in Case 126 (p. 81), and in Case 150 (p. 71) it averaged 48. These suggest that with aortic stenosis the rate tends to be faster, but in the only other patient with aortic stenosis, a man of 63 with high blood pressure, the rate was 34–42 (Case 102, p. 81). All these three had B.B.Bl. also.

Heart rate after exercise. It is generally thought that there is not much change in the heart rate with exercise in cases of complete heart block. Often this is true, at any rate with the gentle exercise they are able to take, and the rise is no more than a few beats a minute. Case 108, for example, showed very little increase even when the auricular rate was doubled. At the other extreme Case 159 showed an increase from 46 to 60 and sometimes to 70 (details are given in the appendix). In congenital complete heart block, where seven cases were tested, I found an average increase from 48 to 55, or to 64 if the rate was calculated from the first quarter-minute only. Even in some of the elderly patients with atherosclerotic complete heart block there may be considerable increases, e.g. in Case 132 the rate was increased from 38 to 58 a minute. Here there was also a change in the type of ventricular complexes (Fig. 5 and 6), but this is, of course, not usual. In the patients with varying degrees of heart block the result of exercise was not easy to foretell. In Case 142 with 2: 1 block the auricular rate

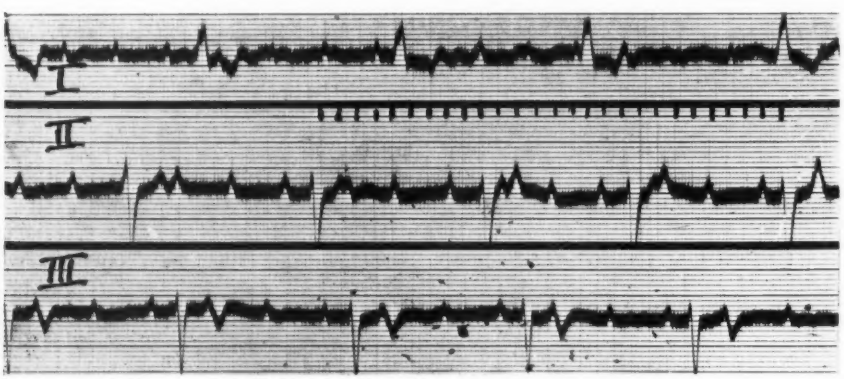

FIG. 5.—Complete heart block with a ventricular rate of 38 from a man, aged 59, who was under regular observation for five years. His B.B. was 200/100 and his W.R. was negative. He had five Stokes-Adams attacks in the first two years, but after taking pot. iod. regularly was three years without an attack except once when he had stopped it for 6 weeks. (Case 132, see also Fig. 6.)

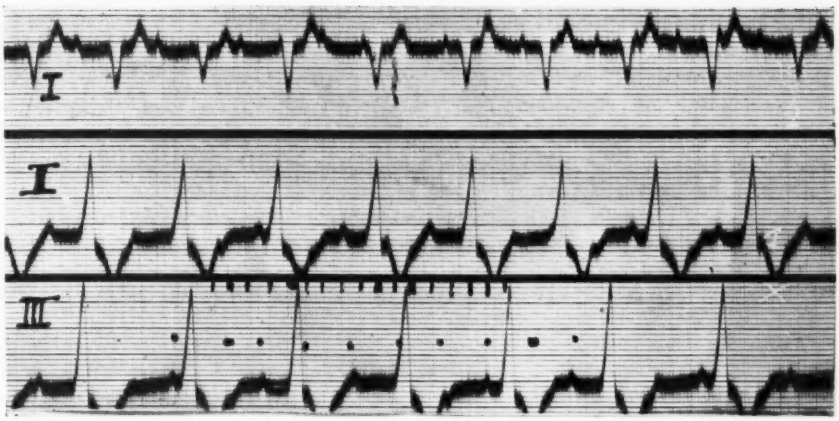

FIG. 6.—Complete heart block from the same case as Fig. 5 with a much faster ventricular rate (58) and a change in the ventricular complexes after exercise. This change was produced on several occasions with exercise (Case 132).

was increased from 70 to 124 while 2 : 1 block was still maintained. In Case 145, 2 : 1 block before exercise changed to complete block after it, but on another day complete block before exercise changed to 4 : 1 block after it.

The blood pressure. The cases were classified as hyperpietic where the systolic pressure was over 160 *and* the diastolic over 100. In these 10, the average blood pressure was 225/108. This is a high pulse pressure as more common averages for ordinary cases of hyperpiesis would be 225/130 or 180/108.

Of the much larger number of cases not falling into this classification of high blood pressure, there were by chance almost exactly equal numbers with a systolic pressure above and below 160. In the former the average figure was 194/81 and in the latter 137/73. Roughly speaking one could say that in half there was a high pulse pressure (about 100) *and* a high systolic pressure with a normal diastolic pressure, and that in half all three were normal, perhaps with a slightly raised pulse pressure. A more detailed analysis, putting the cases into eight equal groups according to the height of their systolic pressure, gave the following average figures: 224/82, 195/86, 187/80, 169/75, 150/81, 141/77, 134/70, and 121/65.

The slow forceful heart beat with its long diastole has been said to produce a high systolic pressure to maintain the circulation adequately and, with more certainty, some fall in the diastolic pressure. This is no doubt part of the explanation but does not seem to be the whole, as half the cases showed so little increase of pulse pressure. Possibly, for this reason, some additional cases should be classified as hyperpietic, because originally they had a higher diastolic pressure, i.e. above 100, but we have no direct evidence of this having happened except perhaps in Case 142 where an early pressure of about 170/97 seemed to have settled at 235/88 six years later.

Probably a more important factor is the atherosclerosis of the aorta that is such a marked feature of many of these cases, though not to a greater extent than in many elderly men. Although several writers have pointed out that as the aorta becomes less elastic a greater pulse pressure is needed to stretch it enough to accommodate the same stoke volume with each heart beat, the significance of this as a cause of a high systolic and high pulse pressure is still insufficiently recognized. It follows from the known physiological facts that the systolic pressure must rise greatly as the elasticity of the aorta diminishes, if the output of the heart is not to fall. This automatically increases the work of the heart. In many elderly patients the diminished output, the increased systolic pressure, and the increased work of the

heart in stretching the aorta are all factors in diminishing the capacity for effort quite apart from any rise of diastolic pressure due to true hyperpiesis which would further accentuate the difficulty.

Bundle branch block. There was some association between bundle branch block (B.B.Bl.) and the lower grades of heart block, nearly 4 per cent of those with latent block having B.B.Bl. There is a closer association between B.B.Bl. and complete heart block. Of the 64 cases, 13 had typical, and 6 others somewhat atypical, B.B.Bl., the combined figures being nearly 30 per cent; in addition another 8 had widened QRS complexes that did not amount to B.B.Bl. In a few cases the B.B.Bl. was irregular, being sometimes present and sometimes absent. In some who had both latent and complete block, there were various findings: B.B.Bl. with C.H.B. and not with N.R. (Case 126, see Table VI), B.B.Bl. with N.R. but not with C.H.B. (Case 111, see p. 84), or B.B.Bl. sometimes with C.H.B. and sometimes with

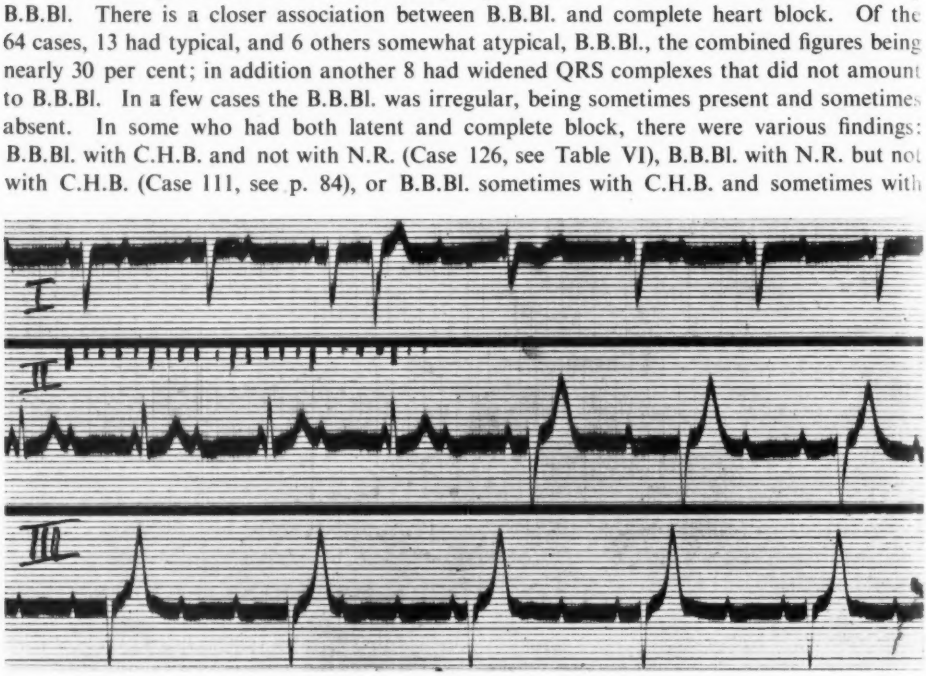

FIG. 7.—Complete and 2 : 1 heart block in a man, aged 60, in whom such changes were frequent. Here the abnormal ventricular complexes came when the block was complete, but this was not always so (Case 131). He had frequent Stokes-Adams attacks and died a year later.

N.R. (Case 102, see p. 81). Fig. 7 illustrates a change in the ventricular complexes when the degree of block changed from 2 : 1 to complete.

All the three patients with aortic stenosis and C.H.B. had B.B.Bl. also.

PERSISTENCE OR CHANGE IN THE DEGREE OF BLOCK

Complete heart block may continue from the time it is first observed until the patient's death. In many cases, however, there are frequent or occasional changes, and different grades of block are observed from time to time. The change may be only to partial block or to latent block, or even to normal rhythm with a normal P–R interval. All cases, except those with congenital complete block, have of course had normal rhythm earlier but this is not included; a few may have had latent block for some time before complete block developed, but the small numbers in whom this sequence of events was observed show that it is not common. It is not easy to classify the different combinations of these changes. This is mainly because of their variety, but partly because the patient is only under close observation for a relatively short time.

Before discussing the changes found in this series of cases the various possibilities will be set out more fully. Complete heart block may be *established* when that rhythm is per-

sistent from the start. It may be *varying*, when complete, partial, or latent block follow one another from time to time. It may be *varying becoming established* when changes are present at first but do not persist. This is a form of progressive heart block but certain cases with a more regular increase in the degree of block have been considered as a special sub-division, *progressive* heart block. It may be *remittent* or *interrupted*, when partial or latent block occasionally interrupts complete block. These remissions may come after a period of years when complete block was present each time the patient was seen and had appeared to be established; in others they are more frequent and it may be difficult to draw the line between this and the *varying* group. All these changes are found in patients who have no Stokes-Adams attacks as well as those who have. In all these groups complete heart block is either the dominant or at least a common rhythm.

There are also cases where complete block is the unusual or temporary rhythm and these can be usefully divided into two groups. It may be *transient*, when it follows cardiac infarction or an acute infection and only lasts during this; or *paroxysmal*, when it occurs without such a known cause. In connection with these cases previous views about paroxysmal heart block will be shortly discussed. The present series of cases will now be considered to see how they fit into the suggested classification.

The details of the changes that were recorded are set out in Table IV. In 29 cases all records taken showed complete heart block, in 3 all showed 2 : 1 block, and in 4 both these rhythms but no other. Another 28 * cases showed complete and/or 2 : 1 block and at other times latent block only, 15 of these having complete block, 2 : 1 block, and latent block at different times.

TABLE IV

GRADES OF HEART BLOCK

	Latent block (total)	Dropped beats (total)	2 : 1 H.B. (total)	C.H.B. (total)	Stokes-Adams attacks	Total number of cases		Corrected figures using clinical evidence also †
C.H.B. only	—	—	—	29	14	29	36	23
2 : 1 H.B. only	—	—	3	—	—	3		2
2 : 1 H.B. and C.H.B. ..	—	—	4	4	4	4		4
C.H.B. and latent block	6	—	—	6	4	6	28	13
C.H.B., 2 : 1 H.B., and latent block	15	6	15	15	6	15		18
2 : 1 H.B. and latent ..	7	3	7	—	5	7		4
Latent block with dropped beats only	29	29	—	—	2	29		
Latent block only ..	140	—	—	—	1	140		
	197	38	29	53	36	233		

† For the rest of the table the grade of heart block had always been proved by graphic records.

Established heart block. At first sight the group of 29 cases with complete heart block (C.H.B.) only might suggest that this rhythm generally persists without change. On examination, however, the proportion had to be reduced somewhat. In 8, few cardiograms were taken because the patients were not seen frequently or did not live long. In 6, faster heart rates of about 60–70 were often observed in the ward, but as they always had C.H.B. when graphic records were taken it is not known if they changed to normal rhythm or to latent block or sometimes to 2 : 1 block. All 6 had Stokes-Adams attacks. In 5 of them the changes

* Case 58, included in my previous paper as latent block only because this was the only rhythm proved graphically, has here been accepted as complete block also since the clinical evidence for a higher grade of block was good.

were frequent; and 3 of these died in less than a year. In the sixth the changes were frequent for three months but in the last six months of his life the rhythm seemed always C.H.B. and he had no more Stokes-Adams attacks.

In the remaining 15, frequent cardiograms were taken more or less regularly, and so far as we know the block was always complete at all other times as well. Of these, 8 died after 1–7 years, and 7 were alive when last heard of after 3–20 years. The classification of these cases would, therefore, be: established 15; varying 5; and varying becoming established 1.

Varying heart block. There were 4 cases with complete and 2 : 1 block. In 3 the changes were frequent but 2 were not under observation for very long; and in 1, the rhythm was generally 2 : 1 during the first few months but later became established C.H.B. for 12 years (varying, 3; varying becoming established, 1).

The 28 patients with complete, partial, and/or latent block must next be considered, and some details are given in Table V. Not all of them, however, are to be classified as varying, in spite of the three rhythms being present: nearly half (13 out of 28) fell into this group and there was little to say except that the different grades of heart block were present from time to time and one had not much idea what rhythm would be found at the next visit. These changes were of all types from Case 109 (p. 84) where the only cardiogram taken showed 2 : 1 block, dropped beats, and latent block in the three leads, or Case 112 who showed these three rhythms on the same day but generally had a slow heart (2 : 1 or complete block), to Case 107.

Case 107. A woman, aged 42, had rheumatic mitral disease without much evidence of stenosis. Her symptoms started with a Stokes-Adams attack and after many attacks she died in one in the

TABLE V

PATIENTS WITH LATENT HEART BLOCK AND HIGHER GRADES

Case No.	Sex and Age D=Died n.t.=not traced	Aetiology	Wassermann reaction	P–R interval with		Complete heart block	Stokes-Adams attacks	Bundle branch block
				latent block	2 : 1 block			
101	m. 57 D	Sy.; B.P. 250/130 (see p. 83)	+	0·17	0·17	+	—	—
102	m. 63–67 D	Ath. aortic st. (see p. 81)	—	0·22	—	+	—	B.B.Bl.
103	f. 66 D	B.P. 250/115 (see p. 81)	—	0·21*	0·21	+	—	—
104	m. 64–69	Heart + (W.R.+)	+	0·34*	0·31	+	—	—
105	m. 67–75	Heart + (see p. 82)	—	0·33	0·30	+	St.A.	—
106	m. 64–65	Heart failure (see p. 82)		0·20	0·19	+	—	—
107	f. 42–47 D	Rh. mitral st. (see p. 80)	—	0·28* / 0·32	0·24 / 0·36	+	St.A.	—
108	m. 34–35 D	Sy. (see p. 81)	(+)	0·22	0·22	+	St.A.	B.B.Bl.
109	m. 52 n.t.	Heart failure (see p. 84)		0·17*	0·19	—	—	—
110	f. 62–63 D	Cor. atheroma		0·24*	0·24	+	St.A.	—
111	m. 73–76 D	Heart + (see p. 84)	—	0·28	—	+	St.A.	—
112	f. 72 D	B.P. 200/100	—	0·29*	0·28	—	—	—
113	f. 29–43	Rh. aortic inc. (see p. 83)	—	0·17*	0·19	+	—	—
114	m. 51 n.t.	Heart + (see p. 81)		0·21†	—	+	—	B.B.Bl.†
115	m. 66–67 D	Heart failure (see p. 85)	—	0·20	0·19	—	St.A.	B.B.Bl.
116	m. 66–67 D	Heart +		0·24	0·24	—	St.A.	—
117	m. 56 D	Heart +		—*	0·26	—	—	—
118	f. 61–64	Cor. ath. (see p. 81)	—	0·24	—	+	St.A.	—
119	m. 49–54	Cor. ath. (Sy.)	—	0·44*	0·44	+	—	—
120	m. 58–60 D	B.P. 210/130 (see p. 81)	+	0·22	—	+	St.A.	B.B.Bl.
121	m. 68 n.t.	Cor. ath.; B.P. 210/90	—	0·26	0·25	—	St.A.	—
122	m. 74–78 D	Heart +	—	0·22	—	+	St.A.	B.B.Bl.
123	m. 46 D	Sy. aortic incomp. (see p. 90)	—	0·32	0·32	+	St.A.	—
124	f. 65–67	B.P. 230/100 (see p. 81)	—	0·28	0·29	+	—	—
125	f. 58–68	B.P. 240/115 (see p. 82)	—	0·23	0·42	+	—	—
126	m. 43–48 D	Ath. aortic st. and inc. (see p. 81)	—	0·25	0·36	+	St.A.	B.B.Bl.
127	m. 61–62	Heart + (see p. 82)		0·24	0·24	+	St.A.	—
58	m. 76–79 D	Heart + ; B.P. 280/100	+	0·36	—	—	St.A.	—

* Dropped beats also at other times.

† Nine years before.

fifth year. During this time she most often had 2 : 1 heart block, but sometimes 2 : 1 changing to latent, or latent block alone, and much less often dropped beats or complete heart block.

Adding these 13 to the 8 already classified as *varying* makes 21 in this group. The remaining 15 of the 28 will be discussed under the following headings though not all are finally accepted in these groups: progressive heart block (7 cases), remittent heart block (5 cases), and temporary heart block (3 cases: 1 transient and 2 paroxysmal).

Progressive heart block (*7 cases*). Here there was some evidence that the grade of block was progressive and finally became complete. On theoretical grounds this might be expected to be more common or almost the rule, but it does not appear to be so. Possibly it may occur more often as a short transient stage when the patient is not under observation, but beyond the cases to be quoted we have no evidence of this.

Only in 3 of the 7 was the first rhythm latent heart block.

Case 114. A P–R interval of 0·21 sec. with bundle branch block was recorded in 1929 in a man, aged 42; in 1938 he began to have attacks of giddiness for the first time and was found to have complete heart block, still with B.B.Bl.; nothing was known about his rhythm in the 9 years between.

Case 118. A woman, aged 60, who had had angina for 2 years and a stroke 5 years before, had a P–R interval of 0·23 sec.; a year later she was found to have complete heart block which persisted for the two years she was under observation; six weeks before this she had her first real Stokes-Adams attack so probably this was the time when her block became complete.

Case 120. A man, aged 58, with high blood pressure was observed regularly for a month with a P–R interval of 0·22 sec.; a year later he was seen with complete block and congestive failure from which he died eight months later; he had no Stokes-Adams attack till just before his death and there was no clinical evidence of when the change of rhythm took place.

Apart from the earlier observation of latent block these 3 might all be classed as established C.H.B. In the next two there was varying partial block as the first finding, and later one had varying and one established C.H.B. Case 108 seems a good example of progressive heart block, but he started with Stokes-Adams attacks six months before his admission to hospital, so that probably he should be classed as varying becoming established. The last should certainly be classified as varying but was interesting from the progressive change in the P–R interval when there was a A-V conduction.

Case 103. In a woman, aged 66, with high blood pressure, dropped beats without progressive lengthening (P–R interval 0·21 sec.) were recorded when she was first seen and she died five months later, her rhythm during the last few months varying between 2 : 1 and complete block.

Case 124. In a woman, aged 65, with high blood pressure, partial block (dropped beats or sometimes 2 : 1 block) was always present during the first six months observation after which she always had complete block; her symptoms of dyspnœa and palpitation were much the same during the three years.

Case 108. A man, aged 34, always had a heart rate of about 66 and a P–R interval of 0·21 sec. for the first fourteen days in hospital. His heart rate then became slower and irregular (generally 40–48) and records showed latent and 2 : 1 block and a more deeply inverted T III. After six days his heart rate fell to 26–36 and apparently complete block became his usual rhythm after this. He was two more months in hospital and died suddenly eight months later.

Case 126. A man, aged 43, with aortic stenosis and incompetence of unproved ætiology, had complete heart block the first time he was seen, but the next time latent block and bundle branch block: and the figures in Table VI show that if the occasions with complete block were omitted there was a progressive lengthening of the P–R interval from 0·24 to 0·41 sec. during the course of three years. He died with congestive heart failure in the fourth year. There was no other case at all comparable with this.

Remittent complete heart block (C.H.B. interrupted generally by latent block (5 cases)).

Case 102. A man, aged 63, with atheroma and aortic stenosis was seen most months for three years and his heart rate was always between 30 and 44 (in cardiograms, C.H.B. with V. 36 to 44) except once during the first year when he had a heart rate of 76 with a P–R interval of 0·22 sec.

TABLE VI.—UNUSUAL PROGRESSIVE LENGTHENING OF P–R INTERVAL

Date	Rhythm	Bundle Branch Block	Heart rate		P–R interval
			Aur.	Vent.	
22/2/24	C.H.B.	B.B.Bl.	100	48	—
9/5/24	Latent B.	N	66	66	0·24
6/6/24	Latent B.	N	50–60	50–60	0·26
10/10/24 ..	2 : 1 B.	N	76	38	0·32
13/3/25	2 : 1 B.	N	86	43	0·32
5/6/25	2 : 1 B.	N	76	38	0·35
23/10/25 ..	C.H.B.	B.B.Bl.	86	39	—
12/2/26	2 : 1 B.	N	80	40	0·41
11/3/27	C.H.B.	B.B.Bl.	110	55	—
27/9/27	(C.H.B.)	B.B.Bl.	76–81	38	(0·48)*
15/6/28	C.H.B.	B.B.Bl.	62	42	—

* Some of this was certainly C.H.B.: some looked like 2 : 1 block with a P–R interval of 0·48 sec., but it might have been chance with the auricular almost exactly twice the ventricular rate.

Case 125. This woman of 58 with high blood pressure was seen regularly for eleven years and always had complete block at each visit, except once in the sixth year when she was seen with normal rhythm at a rate of 60 (to 78) with a P–R interval of 0·23 sec. (Fig. 8), and once in the seventh year when she had a record of 2 : 1 and 3 : 1 block (P–R, 0·40 and 0·26 sec. respectively).

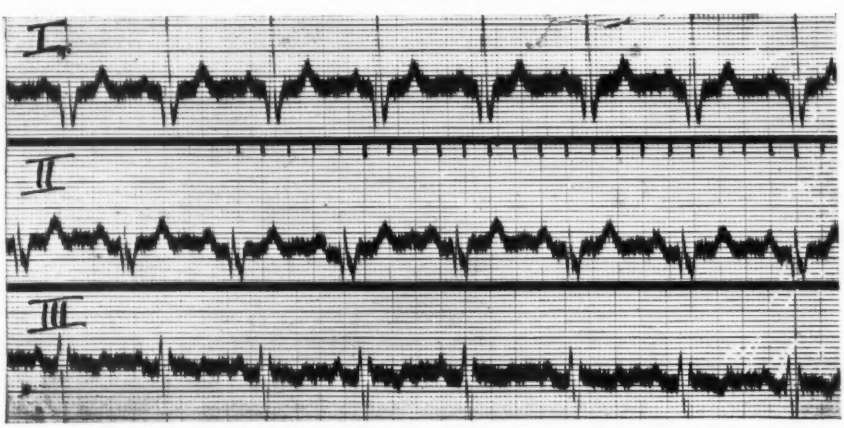

FIG. 8.—Sinus rhythm with latent block, P–R 0·23 sec., interrupting complete heart block that had been established for six years (Case 125).

Case 106. This man of 64 attended regularly for a year and always had complete block except once when latent and 2 : 1 block were both observed.

Case 127. This man of 61 was seen regularly for about 18 months and generally had 2 : 1 or complete block, but on two occasions his heart rate changed from about 40–44 to 60–78 when he had normal rhythm with a P–R interval of 0·24 sec.

Case 105. The last case was less striking, and might have been included on varying. A man, aged 64, always had complete heart block when in hospital in 1931. In 1934, when followed up, he was seen with 2 : 1 block (rate 32; P–R 0·30 sec.) and with latent block (rate 52; P–R 0·33 sec.). In 1939, when he was in hospital again with carcinoma of the penis, he had complete block.

Dividing up the progressive group, where the cases could generally be classified in one of the larger groups, and omitting those where the period of observation was only short, the distribution was as follows:

Established heart block	18 cases
Remittent heart block	5 cases
Varying, latent, or partial, becoming established	7 cases
Varying heart block	23 cases
Paroxysmal and transient block	3 cases

Paroxysmal Heart Block

Before discussing paroxysmal heart block, previous papers on the subject will be considered very shortly. After the case of Hay (1906) with 2 : 1 heart block and a normal a–c interval, Gossage (1909) described a case with intermittent complete block and a normal P–R interval at other times. In the well-known case of Starling (1921) Stokes-Adams attacks could be induced by swallowing though the P–R intervals were normal, but later complete heart blocks became established. Carter and Dieuaide (1923) collected 8 such cases and added one of theirs where there were repititive Stokes-Adams attacks with complete block just after, and a normal conduction time between the attacks: they called it recurrent or intermittent heart block.

Comeau (1937) accepted 12 of 20 possible reported cases as proved examples of paroxysmal heart block and added 1 of his own; most had Stokes-Adams attacks but it was not an essential feature. The same year Gilchrist (1937) said he had seen 8 cases where the sequence of events was normal rhythm, Stoke-Adams attack, complete heart block (for minutes or hours), and then again normal rhythm with a normal P–R interval; he gave details of 2 of the cases, one proved electrocardiographically, and the other by a heart rate of 35 and improvement with ephedrine. Paul White (1937) speaks of cases of heart block as temporary and functional or permanent and organic, but this does not seem to be a sufficient division. Other cases have been mentioned in the paper of Laurence and Forbes (1944). The terms paroxysmal, intermittent, and recurrent have been used indiscriminately for this condition. The former seems the most suitable term for this clinical picture, partly because it is already used for paroxysmal tachycardia and paroxysmal auricular fibrillation.

It has generally been limited to cases where the P–R interval was normal in between, but this prevents such a diagnosis being made precisely in many cases when no graphic record can be obtained of the usual rhythm with a normal heart rate. I suggest that paroxysmal heart block should be used as a clinical term when a normal heart rate with or without latent block is the usual rhythm and heart block the occasional finding. The two groups could be distinguished as paroxysmal heart block (complete) when the P–R interval is normal in the interval, and paroxysmal complete heart block, when there is latent block in the interval. This distinction is of practical importance, for the diagnosis of some Stokes-Adams attacks will be missed unless the existence of paroxysmal heart block (complete) is more widely recognized, because the normal P–R interval found in between will lead to the false assumption that the syncopal or epileptiform attack can not have been of the Stokes-Adams type. Dr. Suzman's case at the end of the appendix is a good illustration of this difficulty.

The term transient heart block could be used when there is a known temporary cause, such as infection or immediately after cardiac infarction.

Transient heart block. One patient fell into this group, as she had temporary block due to an acute infection.

Case 113. A woman, aged 28, already had aortic incompetence and in her third attack of rheumatic fever developed complete block which lasted for a week and was followed by partial block, sometimes with dropped beats and sometimes 2 : 1, this persisting for three months. Full details have been published (Campbell, 1931). In spite of this she made an excellent recovery and is still in good health 14 years later with a normal P–R interval. In this time there has been very little change, and she is able to do a fair amount of work running her father's house.

Paroxysmal heart block. Two patients were included in this group without any doubt.

Case 101. A man, aged 57, with a blood pressure of 250/130 was in hospital with symptoms suggestive of neuro-syphilis and a stroke four years before. He was found with complete heart block which changed sometimes to 2 : 1 and sometimes to occasional ventricular responses with a P–R interval of 0·17 sec. After two weeks the block disappeared and for the rest of the time in hospital he had normal rhythm with the same length of P–R interval. He had a large heart, a strongly positive Wassermann reaction, and mental symptoms suggestive of general paralysis, and unfortunately he was lost sight of as it was necessary for him to be transferred to a mental hospital.

Case 111. A man, aged 73, with high blood pressure, was admitted with Stokes-Adams attacks. He was found to have complete heart block which persisted for four weeks. It then changed to sinus rhythm with bundle branch block and a P–R interval of 0·27 sec., and this persisted for the next month in hospital, and was still present when he was seen a year later. Such an occurrence is, of course, not uncommon where complete block occurs temporarily after a coronary thrombosis, but there was no evidence of this in his case.

With the classification proposed this would be paroxysmal complete heart block, as latent block was present when he had sinus rhythm, and Case 101 and Dr. Suzman's case in the appendix would be paroxysmal heart block (complete).

There are four other cases that must be considered shortly here as they all had heart block (3 with complete and 1 with 2: 1 heart block) and at other times sinus rhythum with a P–R interval that was nearly or quite normal.

Case 106 (p. 82) has been described as remittent because complete block was always found except once when he had 2 : 1 block (P–R, 0·19 sec.) and latent block (P–R, 0·20 sec.).

Case 115. This man, aged 66, was included as varying heart block because there were frequent proved changes of 2 : 1 (P–R, 0·19) and latent block (P–R, 0·20 sec.) and presumed complete block as he started with a Stokes-Adams attack and had several others.

Case 109. This man, of 52, was also included as varying, since 2 : 1 and dropped beats and latent block with a P–R interval of 0·17 sec. were all observed in quick succession: his after history is unfortunately not known and his heart failure may have been due to an acute infection or to infective endocarditis.

Finally Case 103 was included as progressive (partial to varying), but if a P–R interval of 0·21 sec. in an elderly woman may be regarded as normal she could also be regarded as paroxysmal heart block in the sense in which this has generally been used. These last two patients were included by Laurence (1944) being quoted from Table III of an earlier paper (Campbell, 1943*b*).

2 : 1 Heart Block

This is a much less stable rhythm than complete heart block. It must be very rare for it to persist without changes for long periods, though in a few patients it seems the dominant rhythm for some time, occasionally even for years. There were 4 cases with 2 : 1 and complete heart block. In 2 of these frequent changes were observed during the 2 and 5 years respectively that they lived. In the third the block was always, I think, 2 : 1 during the month he was in hospital except once when the rare 3 : 1 rhythm was observed, probably due to atropine. Soon after it became complete and so far as I know this continued for the remaining 12 years of his life, except once in the second year when 2 : 1 block was again recorded (Case 142, see appendix). The fourth has been traced recently after six years (Case 143).

Case 143. A man, aged 38, became short of breath rather suddenly and had at first some minor Stokes-Adams attacks. When seen recently and at the first visit 2 : 1, 3 : 1, and complete block were all recorded: he was quite familiar with the change, which took place frequently, but it seemed that 2 : 1 had remained his predominant rhythm during all the period and that the other rhythms were only present for a short part of the time.

Of the 3 cases where only 2 : 1 block was recorded, none had cardiograms frequently, but in 2 this rhythm seemed persistent and in 1 a heart rate of 70 was observed clinically during the four to six weeks in hospital.

There were also 7 cases where 2 : 1 and latent block were the only rhythms recorded, but in at least 3 of these there was good clinical evidence of complete block and as 5 of the 7 had Stokes-Adams attacks it was probably present in more than this.

3 : 1 *heart block*. This is a much less common rhythm and I have never seen it persist. It is seen most often as a change from 2 : 1 heart block as in Fig. 9.

The P–R interval in 2 : 1 *heart block*. This should have been dealt with in the paper on partial heart block (Campbell, 1943*b*), but was only referred to incidentally when patients had this rhythm and dropped beats as well. It was generally of about the same length as the

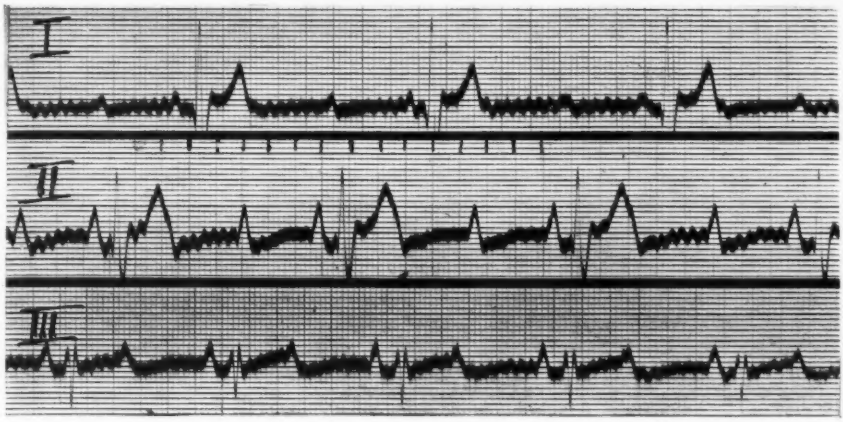

FIG. 9.—3 : 1 heart block in a man who at that time generally had 2 : 1 heart block (as in lead III) and for twelve years afterwards had complete heart block (Case 142). There was little if any change in the P–R interval, which was about 0·21 sec.

first P–R interval with a response. There were 29 cases with records of 2 : 1 block and the P–R intervals were as follows:

Less than 0·20 secs., 8 cases; 0·21–0·22 sec., 4 cases; 0·23–0·24 sec., 5 cases; 0·25–0·26 sec., 2 cases; 0·27–0·32 sec., 7 cases; 0·36, 0·40, and 0·42 sec., 3 cases.

I was surprised that in nearly one third of the cases the P–R interval was normal and sometimes as short as 0·16 sec. In 3 of the 7 cases where 2 : 1 block was the only rhythm observed the P–R interval was normal and only in 2 of the 7 was it over 0·24 sec. The average for all the cases was 0·254 sec. This is identical with figure found for the P–R interval with latent block in cases that had 2 : 1 or complete block also (Campbell, 1943*c*, Table IV). The spread was rather wider as shown by the quantities, 0·19 and 0·30 sec. against 0·215 and 0·270 sec. The fact that the increase in the P–R interval is small or absent in so many cases of 2 : 1 heart block shows again that a defect of excitability or some other factor as well as a defect of conductivity is needed to explain the observed facts.

PROGNOSIS

Two thirds of the cases have died and one third were still alive when last seen after an average period of 6 years. Of the 64 cases, 14 were under observation for a short period only, so have been omitted, leaving 50 for consideration. Of these, 34 died, 10 in less than a year, 9 in the second year, 14 in from 2–6 years, and 1 (Case 142, appendix) after 12 years. The average period of survival was 2·5 years.

Of the 16 who were still alive, 2 were under observation for more than one but less than two years, 8 for from 2–6 years, and 6 for 7, 7, 8, 11, 14, and 20 years respectively. The average period of survival was 6 years. Possibly the last two (Cases 113 and 140, see appendix) should be omitted as the first had transient block during acute rheumatism and the second may have had congenital block though this was not the diagnosis made at the time: if so, the average period would be reduced to 4·4 years. These results are shown in Table VII.

TABLE VII

LENGTH OF LIFE AFTER OBSERVATION OF HEART BLOCK

Length of period	Less than 1 year	1–2 years	2–3 years	3–4 years	4–5 years	5–6 years	6–9 years	10 years or more
No. of patients who have died	10	9	3	4	3	4	0	1
No. of patients who are alive	0	2	3	3	1	1	3	3

The figures agree surprisingly closely with those of Graybiel and White (1936), which suggests that the series are large enough to give an accurate picture. They found that 49 patients had died after an average period of under 3 years, and that 17 were alive after an average period of just under 7 years.

As a contrast the average duration of life in my 10 congenital cases is 22 years, and 7 of the 10 are known to be still living.

From such figures one can calculate the expectation of life if one assumes that surviving patients continue to die at the same rate as those who have died already—probably a pessimistic assumption. If x per cent are alive after an average period of b years, and $100-x$ per cent have died after an average period of a years, $100-x^2/100$ will have died after 2 a years and $\frac{100a-ax+(x-x^2/100)(a+b)}{100-x^2/100}$ will be an approximation to the average duration of life for all cases.

This gives 4·6 years as the expectation of life for complete heart block calculated from my figures and 4·5 years from those of Paul White and Graybiel (1936). The prognosis is better than that for bundle branch block, 3 years, but worse than that for a recent cardiac infarct (excluding deaths in the first six weeks) where it was 6·5 years, these figures having been calculated in the same way from my own cases.

Changes of rhythm did not seem to affect the prognosis unless these were associated with Stokes-Adams attacks. Thus there was no difference as regards prognosis between those having complete heart block *with* latent block and those having complete heart block *without* latent block. If Stokes-Adams attacks are more frequent with changes in the degree of block, a worse prognosis might have been expected. The apparent anomaly might be because there were some cases without proved latent block who had nevertheless changes of rhythm of other types or changes observed clinically but not recorded. The 11 cases of this type were, therefore, transferred and the two groups were analysed again as regards prognosis. But there was still no great difference (see later).

Undoubtedly the prognosis was specially bad in the group with frequent changes of rhythm and Stokes-Adams attacks when they were first seen, but this happened to be balanced by the man who lived 12 years with Stokes-Adams attacks, although at first he had frequent changes of rhythm, and by other similar cases.

There were 36 cases having heart block *without* latent block; 25 of these were under observation for a reasonably long period. Eighteen of these died and 7 were still alive when last heard of. Ten died in less than 2 years; 7 in from 2–6 years; and 1 after 12 years; the average period of survival being 2·7 years. Of the 7 who were still alive, 4 were under observation for from 2–6 years, and 3 for 7, 7, and 20 years respectively; the average period was 4·5 years or 7 years if the last exceptional case was included.

There were 28 cases having heart block *with* latent block; 25 of these were under observation for a reasonably long period. Sixteen of these died and 9 were still alive when last heard of. Nine died in less than 2 years and 7 in from 2–6 years, the average period of survival being 2·3 years; this was a little shorter than in the group without latent block but the difference was not significant. Of the 9 who were still alive, 2 were under observation for more than 1 year, 4 for from 2–6 years, and 3 for 8, 11, and 14 years respectively; the average period was just over 6 years, or 4·3 years if the last case be excluded. Again the period was a little shorter but the difference was not significant.

If the patients were divided into two groups—those known to have different grades of heart block and those only known to have complete block—by the transfer of the 11 cases referred to, the difference between the two groups were still slight.

There were 25 cases *without change of rhythm*, 8 of whom were not traced: 11 died (5 in less than 2 years, and 6 in from 2–6 years), the average period of survival being 2·5 years; and 6 were still alive (4 after 2–6 years, 1 after 7 years, and 1 after 20 years), the average period being 7·0 years, or 4·3 years if the last case be excluded.

There were 39 cases *with change of rhythm*, 6 of whom were not traced: 23 died (14 in less than 2 years, 8 in 2–6 years, and 1 in 12 years), the average period of survival being 2·7 years (2·3 years without the last case); 10 were still alive (2 after 1 year, 4 after 2–6 years, and 4 after 7, 8, 11, and 14 years), the average period being 4·4 years, or 5·4 years if the last case be excluded.

Incidence of Stokes-Adams Attacks

Just over half the patients with complete and/or 2 : 1 heart block had Stokes-Adams attacks—33 out of 64. Originally, as the relationship with latent block was being specially investigated, they were divided into two groups—those with and those without recorded latent block—and rather surprisingly in view of the reputed association of Stokes-Adams attacks with a changing degree of block there was no significant difference in the incidence of Stokes-Adams attacks (53 against 50 per cent). But there is no great danger in the change from latent block to dropped beats or to 2 : 1 block when no new centre has to take control, and it is only the change from one or other of these rhythms to idiopathic ventricular rhythm that is attended by the risk of a Stokes-Adams attack.

Among the group without recorded latent block there were 4 with 2 : 1 block, and 7 with faster rates that did not chance to be graphically recorded. Here the incidence of Stokes-Adams attacks were specially high, 10 of these 11 cases, possibly because they were in hospital for their Stokes-Adams attacks just at a time when these were most frequent owing to the frequent changes of rhythm. If these 11 cases were moved, 25 of the 39 with known change of rhythm had Stokes-Adams attacks (64 per cent), and only 8 of the 25 with no known change from their complete heart block (32 per cent). This is not meant to imply that even in these last there was not a change of rhythm at the time of their Stokes-Adams attacks, but that no such changes were noted at other times when they were under observation, and that the precise behaviour of the heart with their attacks was not known. There were several cases where Stokes-Adams attacks were more frequent at or near the start and the patient " grew out " of them. This may be partly due to the habituation to the slow ventricular rate and to the ventricle having become accustomed to its responsibility for initiating and maintaining the heart beat. For example, Case 136 had two Stokes-Adams attacks when 66; when 68 he was in hospital and though during the first two weeks his heart rate varied between 40 and 72 he had no more than attacks of faintness; there was little change when he was 70 and he had had no real Stokes-Adams attacks since the first two. Case 143 (p. 84) gave a very similar history. Case 145 had frequent attacks all through the day for ten days at the start when he was 62; he lived 6 years after this and the attacks were not very frequent though they continued. In Case 147 the first attack was the only typical one, though he had attacks of faintness when things went black for the remaining nine months of his life.

Prognosis with Stokes-Adams Attacks

As some of the cases were not traced there were 50 available for assessing the prognosis. It was considerably worse when there were Stokes-Adams attacks, 80 per cent of these patients having died against 50 per cent of those without such attacks.

TABLE VIII

STOKES-ADAMS ATTACKS AND PROGNOSIS

	Total	Alive when last heard of	Dead	Not traced
Patients with Stoke-Adams attacks	33	6	24	3
Patients without Stoke-Adams attacks	31	10	10	11

Unfortunately there were far more who were not traced among the patients without such attacks, and if all these were included the percentage figures were:

With Stokes-Adams attacks .. .. Known to be alive, 19 per cent; dead, 73 per cent.
Without Stokes-Adams attacks .. .. Known to be alive, 33 per cent; dead, 33 per cent.

Although more of the patients had died while under observation the length of time before death was not significantly different, the average for both groups being between 2 and 2·5 years. This apparent anomaly is explained by the fact that there are more of the patients without Stokes-Adams attacks still surviving, who will later increase the average duration of life considerably.

There was no noticeable difference in any of these findings between the patients who had and those who had not latent heart block. All the figures have therefore been combined.

Method of dying. The great danger of sudden death in patients with Stokes-Adams attacks has of course been emphasized for a long time. This was again confirmed in the present series, but I was surprised to find that there was only one record of sudden death in the patients with complete heart block where a history of Stokes-Adams attacks had not been noted. No doubt patients with or without previous heart block may sometimes die in their first Stokes-Adams attacks and so come under the notice of the pathologist rather than of the clinician, but in this series only one patient seen with heart block without any evidence of Stokes-Adams attacks is known to have died suddenly. There were a few without Stokes-Adams attacks when first seen who developed this later and so lost their immunity from sudden death, but even so the point seems of considerable value for prognosis.

Of the 24 patients with Stokes-Adams attacks who died, no less than 14 died suddenly (almost certainly in attacks), 3 died with heart failure, and in 7 the method of death was not known.

Of the 10 patients without Stokes-Adams attacks known to be dead, 1 died suddenly, 3 had congestive heart failure, 1 left ventricular failure, 1 broncho-pneumonia, 1 cerebral hæmorrhage, 1 intestinal obstruction, and in 2 the method of death was not known.

One practical and valuable conclusion from these facts is that the patient who has not had a Stokes-Adams attack in the first month or so of his heart block is not very likely to develop such attacks, and the longer the time that passes without them, the more certain one can be that there is small risk of his dying suddenly.

Summary and Conclusions

Complete heart block is most often seen in men in the seventh decade with enlarged hearts and atherosclerosis but no other evidence of gross heart disease. Four-fifths of our patients were men. Most (45 per cent) were between 60 and 69, and 84 per cent were over 50 years of age at the onset of complete block.

Syphilitic and rheumatic heart disease were between them responsible for only just over 10 per cent of the cases. Other myocardial disease was responsible in 75 per cent, or in 86 per cent if the group of congenital cases was excluded, this being the second commonest cause (13 per cent). Cardiac enlargement with no other signs than atherosclerosis of the aorta and often of peripheral arteries was the evidence of myocardial disease in nearly half these cases, high blood pressure, angina pectoris, or congestive failure being present in the other half. In the 10 cases with high blood pressure the average figure was 225/108. In the others the systolic pressure was above and below 160 in equal numbers and the average figures for these two groups were 194/81 and 137/73. Thus, in the latter the pulse pressure was only slightly raised, but in the former and in those with high blood pressure, the pulse pressure averaged 115. The reasons for this have been discussed.

The heart rate was usually between 28 and 40 and averaged just under 35 (excluding congenital cases where it was generally 40–56).

Heart block may be of very varied types: it may be (1) complete; (2) partial; 2 : 1, or more rarely 3 : 1 or 4 : 1; (3) partial with dropped beats only; including regular 4 : 3, 3 : 2, etc. or occasional dropped beats; or (4) latent, with a prolonged P–R interval only.

In addition to this there may be various changes and combinations which can be usefully described as follows. Complete heart block may be *established*, or *varying*, changing to other degrees of block, or *interrupted*. It may also be *transient*, due to a known infection or to a specific episode such as cardiac infarction, or *paroxysmal*, of which there are two varieties *paroxysmal complete heart block* when the usual rhythm is latent heart block, and *paroxysmal heart block* (*complete*), when the usual rhythm shows a normal P–R interval. These varieties and combinations have been discussed.

Prognosis. Complete heart block is a serious lesion, though some patients, especially some of those under 40, live for many years in reasonably good health. There were 50 cases followed for more than 2 years or until their death; 34 were dead after an average period of 2·5 years; 16 were alive after an average period of 6 years or 4·5 years if two exceptional cases were excluded. Of the former, 19 died in less than 2 years, 14 in from 2–6 years, and 1 after 12 years. Of the latter, the period of observation was from 2–6 years in 10, and from 7–20 years in the other 6 cases.

Stokes-Adams attacks. These were present in half the patients with complete heart block. When they were present they were one of the earliest if not the first significant symptom of heart block in three-quarters of the cases. In those without Stokes-Adams attacks, dyspnœa or attacks of faintness or dizziness were the main presenting symptoms.

Stokes-Adams attacks were no more common in those who had *recorded* latent heart block than in those without. A known change of rhythm at times other than those of the attack does make Stokes-Adams more likely but not as much as might be expected (64 against 32 per cent).

The prognosis was considerably worse in those with Stokes-Adams attacks, the results in the patients traced being:

	Alive	Dead
With Stokes-Adams attacks	6	24
Without Stokes-Adams attacks	10	10

The method of dying was even more strikingly different: of the patients with Stokes-Adams attacks, 61 per cent died suddenly, presumably in attacks; of those without Stokes-Adams attacks, only one was known to have died suddenly and 50 per cent died with failure. If when a patient is first seen with complete heart block he has not had a Stokes-Adams attack, the risk of such an attack developing or of his dying suddenly is not great, and with each month that has passed the risk becomes still less.

It is important to realize that Stokes-Adams attacks may occur with paroxysmal heart blocks (complete) and the paroxysms may be of short duration after the attacks and may easily be missed. Otherwise attacks that are true Stokes-Adams attacks will remain unexplained.

I should like to express my thanks again to my colleagues at Guy's Hospital and at the National Hospital for Diseases of the Heart, and especially to Dr. S. Suzman, my clinical assistant, and Mr. F. H. Muir, technician to the Cardiographic Dept., Guy's Hospital, for helping me to keep in touch with so many of these patients.

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Appendix of Case Notes

An Unusual Case of Stokes-Adams Attacks and Paroxysmal Heart Block

Case 123. K. R. had malaria, dysentery, and gonorrhœa in Rhodesia; the last was treated very inadequately and was followed by crippling arthritis. Soon after he began to have attacks of vomiting with blood in the vomit and 7 years later, as this persisted, Sir Arthur Hurst diagnosed gastritis polyposa and arranged for gastrectomy (*Hurst, A. F., and Stokes, A.* (*1926*), *Guy's Hosp. Rept.*, **76,** *351*). So far no mention has been made of his heart but section of some of the larger vessels of the stomach showed arteriosclerotic changes.

In 1923, when he was 34, he had his first syncopal attack and only remembered finding himself on the ground surrounded by servants. In 1927–8 he had 2 or 3 more attacks and in 1929 when on leave had 4 attacks in four months. Nearly all occurred when he was standing and drinking whisky. They were preceded by a momentary feeling of nausea and sometimes, if he was sitting, he had this feeling without any after developments. He never hurt himself in falling and thought he was out for up to a minute. Afterwards he felt unsteady and sometimes vomited a little bile but recovered quickly and could take another drink without effect.

In 1929–34 he had 7 or 8 more attacks. In 1935 he was readmitted to Guy's Hospital for severe arthritis of his spine and large joints. His heart rate was slow, due to varying heart block, most often 2 : 1, sometimes complete or latent. The heart was just enlarged and there was an aortic diastolic murmur. The W.R. was negative. He had no more attacks in hospital but died six months later.

Here, no precise diagnosis of the cause of the syncopal attacks were possible until some years later he developed varying heart block: it is still only a reasonable deduction that they were Stokes-Adams attacks and that he had paroxysmal heart block; it seems that drinking spirits may have been a special provoking cause as swallowing was in the case of Starling (1921).

C.H.B. of Doubtful Actiology: Good Health after 20 *Years*

Case 140. K. H. had her first and only attack of fainting when she was 19, and C.H.B. was diagnosed then. She was seen by me 12 years later, complaining of no more than some breathlessness and giddiness, and she remained under regular observation for 8 years more.

Her heart was considerably enlarged to the left with an apical systolic murmur; her B.P. remained about 180–200/80–90; her W.R. was negative; and there was no evidence of valvular disease. Her heart rate was early always between 30 and 40 and the only other cardiographic abnormality was left ventricular preponderance which remained unchanged. She continue in good health able to do light house work. It has not been possible to trace her as the area where she was last living has been bombed.

She was classified as a rheumatic case because of a history of doubtful rheumatic fever when she was 10 but there was no clinical or radiological evidence of mitral stenosis. On reviewing her case I feel it much more probable that this was a case of congenital heart block that I failed to recognize, even in my recent paper (Campbell, 1943, *a*).

Unusual Length of Life with Stokes-Adams Attacks

Case 142. J. H., a seaman, had no history of rheumatic fever or syphilis, and his W.R. was negative. He always had good health and served in the war 1914–18 without any complaints. In 1919, when 43, he began having attacks in which he went unconscious and fell. At first they were preceded by precordial pain which spread up the left side to the angle of the jaw and this seems to have been most severe with the first attack. His only other symptom was dyspnœa, but he had to give up work because of the frequency of the attacks—at least one a month.

In 1927 Dr. L. Forman wrote to me as follows: " This man was admitted to hospital as an epileptic. During an observed attack he became very pale and his pulse could not be obtained; later it became regular at 20 a minute; and has since been about 45." He was admitted under my care and was found to have 2 : 1 heart block (V, 40–48). His heart was slightly enlarged and his arteries extremely thickened. During a month in hospital he had no attacks. All records showed 2 : 1 block, even when his auricle was quickened to 120 with exercise, except once when he had been given I.A.H. 1/150 grain 4 hourly, enough to produce dryness of the mouth; he then showed 3 : 1 heart block (Fig. 9). Records of his B.P. varied, e.g. 160/90, 180/90, 170/110. A year later he came up with a pulse of 32 due to C.H.B.; his B.P. was 230/120. With these two exceptions there was no change in his condition. The following months he again had 2 : 1 block after which every record for the next 10 years showed complete block.

He was last seen in 1938 and had been frequently an out-patient and three times in hospital. His attacks had continued unchanged though no others were observed. His description of the attacks was very much the same as at the start, but the pain became a more permanent feature again: it has an even more typical anginal spread but he had no regular angina of effort. In his later years the B.P. was generally about 240/90. In some of his more severe attacks he bit his tongue and passed water—episodes which are stated not to occur in Stokes-Adams attacks, but it seems unlikely that this was a case of complete heart block developing in an epileptic and I have a direct observation by a competent observer confirming the absence of the pulse during an attack.

He died suddenly in his sleep in 1939, 20 years after the first attack and 12 years after he came under observation with heart block.

C.H.B. of Unknown Actiology with Relatively Fast Ventricular Rate

Case 159. W. W. complained of nothing till she had three or four fainting attacks when she was 18. Her doctor told her her heart was slow and sent her to hospital when complete heart block with a rate of 40–48 was confirmed. Her heart was a little enlarged with a systolic murmur; nothing else abnormal was found; B.P. 135/70. Her slight dyspnœa improved with symptomatic treatment; her fainting attacks did not recur, and she was working and leading a normal life six years later.

The diagnosis of congenital C.H.B. was excluded because when she had scarlet fever at the age of 11, the three recorded pulse rates were 100, 70, and 90, and there had been no comment on her heart then or when she had pneumonia as a child. She had no family or personal history of rheumatic fever and I did not think she had mitral stenosis though this was difficult to exclude with absolute certainty; nor had she any history of diphtheria. Although she had no signs of congenital syphilis and a negative W.R. she was classified as syphilitic because her father had died of general paralysis, but I feel less certain now that she was not a case of congenital complete heart block.

The effect of exercise was carefully observed on several occasions and she was unusual in that the ventricular rate could be increased to 70 (Fig. 10). The A/V ratio varied on different days but tended to be fairly constant during and after exercise on a particular day (see Table IX, where some rates recorded graphically are given). This relative constancy of the A/V ratio suggests that both were being influenced by the same factor, possibly by the carbon dioxide content of the blood.

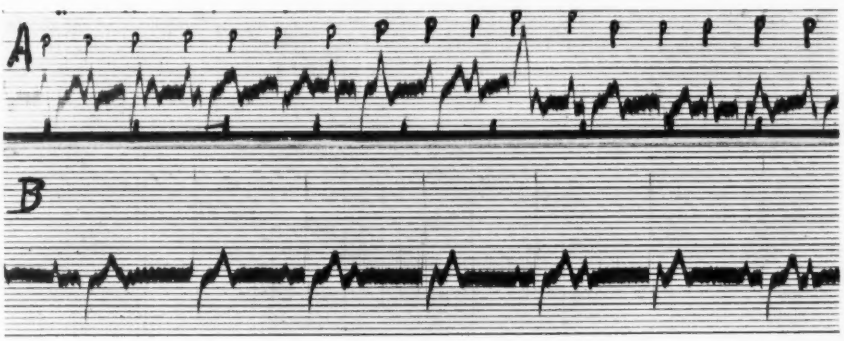

FIG. 10.—Complete heart block of unknown actiology (? congenital syphilitic) with a relatively rapid rate of 48 at rest (B) and a faster rate of 70 after exercise (A) from Case 159. In (A), which is lead I, the P waves have been marked; (B) is lead II. The time marker in (A) has been inked in to indicate seconds.

TABLE IX

AURICULAR AND VENTRICULAR RATE AT REST AND AFTER EXERCISE

Date	Auricle	Ventricle	Ratio
	At rest		
27/5/27	77	49	1·57
21/7/27	74	42	1·78
25/1/28	79	44	1·79
24/5/28	75	48	1·56
	After exercise		
27/5/27	112	70	1·60
,,	78	50	1·56
21/7/27	106	59	1·80
,,	87	48	1·80
,,	76	44	1·72
25/1/28	111	60	1·85
,,	102	50	2·04
24/5/28	110	71	1·55
,,	86	56	1·54
,,	75	48	1·56

Dr. Suzman's Case. Stokes-Adams Attacks with Paroxysmal Heart Block (Complete)

The following patient, notes and records of whom have kindly been lent me by Dr. S. Suzman, had Stokes-Adams attacks due to ventricular standstill without any evidence of heart block except

for a few minutes after a recorded Stokes-Adams attack. She did have bundle branch block but this was not persistent.

In 1937, when she was 64, she had her first attack of gall-stone colic and jaundice. In April 1941 she noticed she was short of breath and in August she became giddy and collapsed. In September, after another collapse, she was admitted to Guy's Hospital and diagnosed by Dr. Suzman as having Stokes-Adams attacks; she had bundle branch block (B.B.Bl.) with a P–R interval of 0·17 sec. Attacks were frequent enough for Dr. Suzman to hope for a graphic record, and after waiting some hours with everything ready he succeeded.

Before the attack there was B.B.Bl. with a normal P–R interval; in the attack there was ventricular standstill for nearly 30 seconds; and after the attack there was probably complete heart block lasting for at least a minute. At this stage no further film was available and the next day there was again B.B.Bl. with a P–R interval of 0·17 sec. She was discharged soon after and during the next month cardiograms taken while she was an out-patient were normal in every way except for rather low T waves, the P–R interval being 0·16 sec.

In January 1942 she was admitted to another hospital, and in view of her recurrent attacks of colic it was decided to remove the gall bladder, and this was followed by a second operation three weeks later for localized peritonitis. She was home well in April. During this stormy period her heart rate was regular and 70–90 a minute, except for some days after her operation, when it rose to 120, and no attention would have been drawn to her heart apart from the recent history of the attacks, which did not recur. Throughout her stay she took ephedrin, 1/2 grain daily. Her heart was enlarged (16·5 cm. m.t.d. in a chest of 26 cm.). The B.P. was 110/70.

A RARE CASE OF COMPLETE HEART BLOCK

BY

KEMAL SARACOGLU

From the International Clinic, Constantinople

Received October 23, 1943.

Complete heart block is not rare, but the case to be described has some very unusual features. A woman, aged 50 years, working as a doctor's servant, first came to us in March, 1937, for intestinal worms. She also complained of anxiety and dyspnœa, but at the first consultation nothing was mentioned about her real illness.

On 23/2/38 she came again for her heart disease. She complained of anxiety, heartache, and dyspnœa. Her illness had begun three months before and she stated that she suffered more during the night than the day. She was married and had borne five children, who had all died during the first days of their life. Her husband had died after a short illness, but she did not know the name of his disease.

At the examination of the patient we observed the following: her face was pale and her

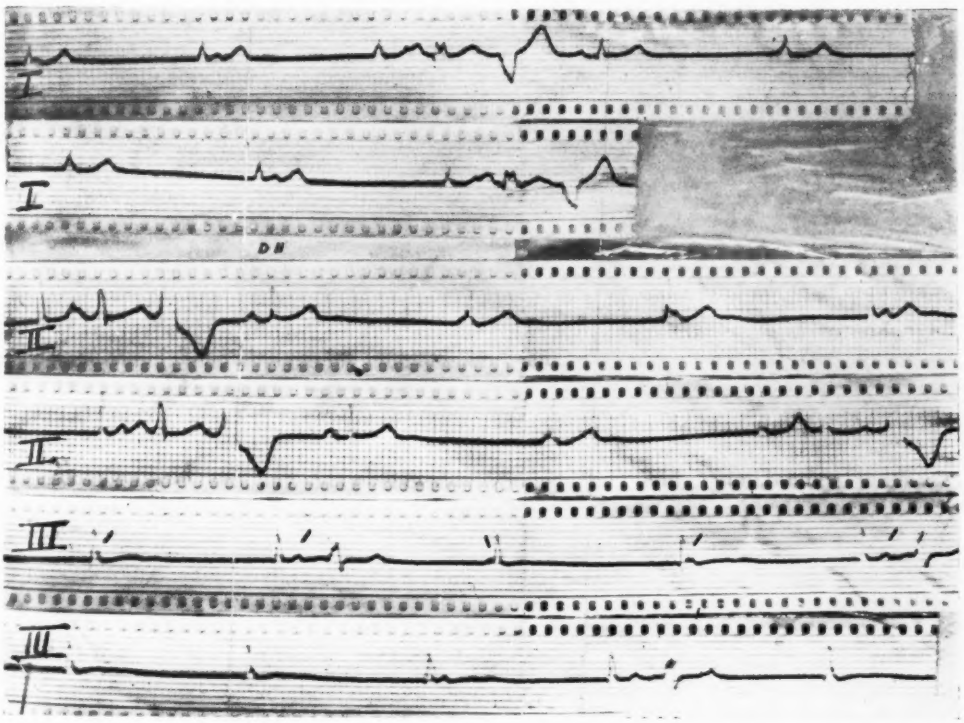

FIG. 1.—Electrocardiograms showing complete heart block with frequent ventricular extrasystoles, and the ventricular rate (about 40) slightly faster than the auricular rate (about 39 a minute).

dyspnœa increased during exertion. Her heart beats were irregular on account of extrasystoles. Her pulse rate was 40. This bradycardia was a serious symptom. On examining her lungs we found some dry rales. Her blood pressure was 200/100 with the Vaquez apparatus. There was nothing important in her other organs. The urine was normal. The Wassermann and Kahn reactions were negative.

The most important findings were in the cardiogram (Fig. 1). Extrasystoles appeared after four or five ventricular beats. The number of the regular beats were 40 a minute. There was an important bradycardia caused by complete heart block, because the auricular beats were independent of the ventricular beats. One point attracted our special attention, namely, that the number of the auricular beats were 39 and the ventricular beats 40 a minute. Indeed, in complete heart block it should be the contrary, with the auricular beats from about 60 to 80 and the ventricular beats about 35 a minute. So a complete heart block was combined with slowness of the auricular rate. What could be the cause of this slowness? The following diseases must be considered: cerebral tumour, meningitis, cerebral commotion, cholæmia, digitalis intoxication, typhoid fever, hypothyroidism, vagotonia, and last, coronary sclerosis. In our case only two things seemed to be possible: (1) vagotonia, or (2) the destruction or degeneration of the Keith-Flack node.

In order to discard the first possibility we injected atropine and adrenaline and afterwards we took a new cardiogram (Fig. 2). These medicaments did not change the slowness of the

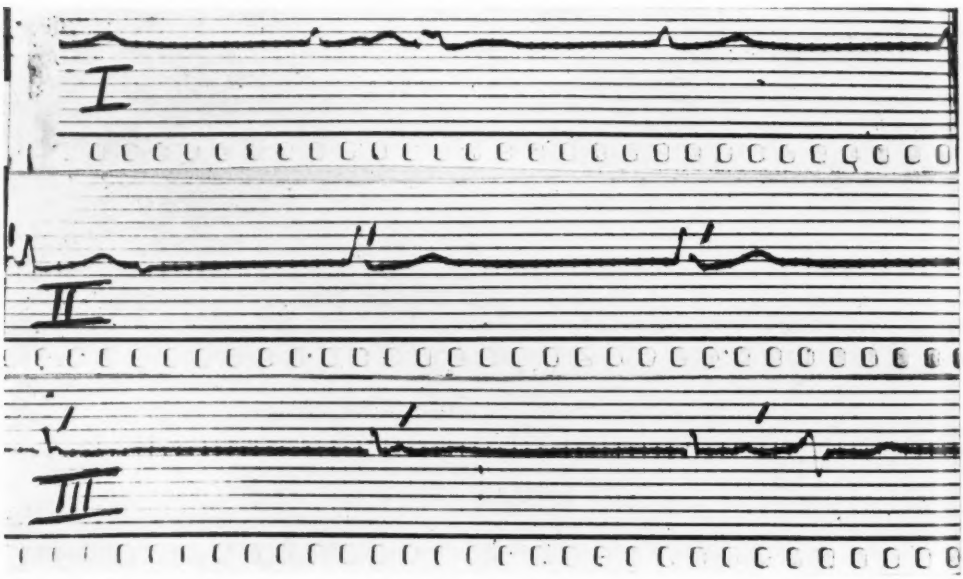

FIG. 2.—Electrocardiograms after the injection of atropine and adrenaline, showing no significant change in the slow auricular rate.

auricular beats. Therefore we decided that it was not the result of vagotonia, but of a coronary sclerosis. The coronary arteries being unable to vascularize the Keith-Flack node, the excitation produced was under the normal rate.

This form of complete heart block with slow auricular beats is very rare. Some parts of the records taken with Siemens apparatus appeared somewhat like fibrillation or nodal rhythm, but the figure as a whole show that it was an example of complete heart block.

Further records were not possible because the patient died. At the section of the body we found a diffuse myocarditis.

SUMMARY

A case of complete heart block with a rare degree of auricular bradycardia is described. The ventricular rate was 40 and the auricular rate 39 a minute.

As the auricular rate was not influenced by atropine or adrenaline it was thought to be due to coronary disease and not to increased vagal tone.

FREDERICK JOHN POYNTON

F. J. Poynton, son of the Rev. F. J. Poynton, rector of Kelston, Bath, was born in 1868 and died on October 29, 1943. He and his elder brother, A. B. Poynton, formerly Master of University College, Oxford, were both educated at Marlborough, in the welfare of which he remained keenly interested. He felt that he owed to one of his form masters there the impulse to take up his active medical career.

He started his medical studies at University College, Bristol, and won a scholarship at St. Mary's Hospital, London, in anatomy and physiology. Qualifying in 1893, he gained first-class honours in the London M.B. the following year, took the M.D. in 1896, and was elected F.R.C.P. in 1903.

In 1900 Poynton became physician to out-patients at the Hospital for Sick Children and in 1903 assistant physician to University College Hospital. His work was done at these two hospitals with which he remained connected till the end of his life, though when he retired from the active staff in 1934, he went back to live near Bath, at Coombe Park.

Poynton will be mainly known for his work with Paine, trying to show that rheumatic fever with chorea was a specific infective disease and that it was caused by the *Diplococcus rheumaticus*. The latter part of this view was never widely confirmed and has not been accepted, and this has perhaps obscured a full appreciation of his other work bearing on acute rheumatism. When the time comes for a full understanding of the cause of acute rheumatism, it may be that his work will be found to have been along the right lines even if there were errors in the bacteriological technique.

His research work may be divided into these main stages. First in papers with Lees (*Medico-Chir. Trans.* , **81,** 419, 1898) and Still (*Trans. Path. Soc.* London, **50,** 324, 1899) and others, he dealt with the dilatation of the heart and the changes in the muscle during rheumatic fever, and with the histology of the rheumatic nodule, arguing from these that rheumatism was of infective origin. There are also statistics of some measurable features of rheumatism in children and good clinical observations—data which have helped to establish modern clinical views.

The second stage, in association with Paine, dealt more directly with the discovery of the *Diplococcus rheumaticus*. His first paper published in 1900 (*Lancet*, **1,** 1352) represented some years intensive search for the infecting organism and results were not published until they had found it in the pericardium, in the heart muscle, in the joints, and in the rheumatic nodule, and until they had reproduced a good imitation of the disease in rabbits (an animal that has unfortunately proved itself a most unreliable guide for human pathology).

In the last group of his publications many of the papers are now of less interest as they were concerned with criticisms that had been made of the *Diplococcus rheumaticus* and with his rather too ardent defence of the work by himself and Paine. There are, however, others of more general clinical interest. One dealt with 52 of his cases of acute rheumatism in children under 5 years, 43 of them having heart disease: the features of rheumatic and pneumonic pericarditis were also contrasted (*Quart. J. Med.*, (1908) **1,** 225). Another on so-called scarlatinal rheumatism brought forward evidence that the disease is identical with ordinary rheumatic fever, having followed and complicated the original scarlet fever—a view which has now gained general acceptance (*Quart. J. Med.*, (1909) **3,** 15). Another discussed his view that malignant endocarditis might sometimes be rheumatic (*Quart. J. Med.*, **5,** 463, 1912) but he failed perhaps to prove that there was not a combination of a terminal infection on an old rheumatic valvu-

litis. Yet this paper does show the importance of rheumatism as a cause of aortic valvular disease which has sometimes been overlooked or minimized.

In 1931, with Bernard Schlesinger, he published a book on *Recent Advances in the Study of Rheumatism*. He also wrote a small book on *Heart Disease and Thoracic Aneurysm* (1907) and with Paine republished many of his papers with the title *Researches on Rheumatism* (1913), a work dedicated to Lees, his friend and teacher. He ended this with a chapter on the prevention of acute rheumatism from which the following quotations are taken. He would, I think, have been pleased with the short review and the joint report on the Care of Rheumatic Children that follow this article.

" The importance of the prevention of an infection which is the great cause of organic heart affections in the young is one that cannot be over-rated; and we believe that definite results will be forthcoming because acute rheumatism is met with much more frequently among the poorer classes and is encouraged by those agencies, which may be summed up in the single word ' poverty.'

" In the great hospitals it is clear that we possess ample means for treating the acute disease and its complications and for advancing its study in many directions. Their value might well be strengthened in the future by the establishment of some special convalescent homes in carefully chosen sites for those recovering from early rheumatism including heart disease and chorea.

" We are convinced that the medical inspection of children in the State schools will in the near future provide us with valuable information upon the influence of school life on chronic rheumatic heart disease and on the relation of out-breaks of acute rheumatism to epidemics of sore throats.

" The education of parents and school teachers in the principal dangers of acute rheumatism might well be carried on by means of simple instructions and lectures. Among such instructions may be suggested the following for rheumatic children.

1. The importance of proper clothing.
2. The care of sore throats.
3. The necessity for attention to ' growing-pains.'
4. The importance of undue nervousness, clumsiness, and night-terrors, as warnings of chorea.
5. Parents should be warned that the early signs of heart disease are few, and that shortness of breath is more often complained of than pain.
6. Much emphasis should be laid upon need for patience when a child is recovering from heart disease.
7. Parents should be told that rheumatism is very liable to recur.

" We believe that more attention might be given to the condition of the tonsils and naso-pharynx in the rheumatic child, and also to the choice of an employment for those who have been damaged by the disease.

" The view that acute rheumatism is an infective disease raises again the important questions of climatic and local surroundings, sanitation, and conditions of housing. The possible relation that it may bear to other infective processes seems worthy of further inquiry.

" It is to *prevention*, then, that we look for some advance from this grievous state of affairs, and to this pressing need that we have ventured to publish this book."

Poynton never became deeply interested in modern cardiology and despite so much devoted and passionate work on the heart in rheumatism he was primarily a children's physician. It was here that he was at his best and his real understanding of the child as well as of its disease ensured his success, which was widely recognized as when he became President of the British Pædiatric Association in 1931.

Poynton was a good teacher and as one writer has said " His mischievous frankness of speech endeared him to students." He was also a good chairman of committees and was Senior Censor of the Royal College of Physicians in 1930.

He was a man with many interests and had been a fine cricketer who played for Somerset from 1891 to 1896; he also played hockey for Middlesex. He had a great love of music and a good tenor voice inherited from his parents. I am indebted to his friend Sir Henry Tidy for permission to quote the following appreciation, published in the *British Medical Journal*.

" Poynton was a character and sometimes appeared almost wishful to be an oddity. He was a countryman, and deliberately retained a countryman's characteristics with a certain scorn of personal appearance.

Short and stocky in build, he was enormously strong. He held the curious record of captaining a first-class county cricket eleven after attaining to the sober dignity of a Fellow of the College of Physicians. His outstanding cricketing feat was achieved in two hours at Hove

when he and Sammy Woods scored 305 runs at top speed for Somerset against Sussex, and he loved to recall the episode. He played hockey for Middlesex and had some skill at real tennis. He had a fine tenor voice and sang for many years in the choir of St. James' Church.

Unfortunately he allowed his professional life to be clouded by what he considered to be the unsympathetic attitude of the profession towards his researches on rheumatic fever, though his resentment never extended to individuals. When speaking in public and at medical meetings he seemed to delight in supporting some illogical standpoint, and while he amused his audience he was not taken very seriously. But when he dealt with a sick child he had a knack of talking spontaneously as if they were equals, and every child felt at ease with him.

He was a delightful personal friend and a great companion on a holiday. He enjoyed every moment, and to the end he retained his power of boyish enjoyment, his sincerity and his honesty and his love of children."

MAURICE CAMPBELL.

THE CARE OF RHEUMATIC CHILDREN

REPORT BY THE CARDIAC SOCIETY AND BRITISH PÆDIATRIC ASSOCIATION

In 1942 the Cardiac Society appointed a small committee to draw up a report and make suggestions about the arrangements that should be made for the care of rheumatic children. When it was learnt that the British Pædiatric Association had appointed a similar committee and that some members were serving on both bodies it was hoped that a joint report might be made. This wish was shared by the British Pædiatric Association and the two committees met again as a joint committee and drew up a report. After various minor modifications the Council of the Cardiac Society and the Executive of the British Pædiatric Association agreed to publish the report that follows. They are greatly indebted to the following members who drew up the original joint report:

F. M. B. Allen (B.P.A.), J. V. Braithwaite (B.P.A.), T. F. Cotton (C.S.), W. Evans (C.S.), C. B. Perry (B.P.A. and C.S.), C. T. Potter (B.P.A.), J. C. Spence (B.P.A.), A. G. Watkins (B.P.A.), K. D. Wilkinson (B.P.A. and C.S.), T. P. Williams (B.P.A.), and B. Schlesinger (C.S.) (who was prevented from agreeing to the final draft owing to absence on active service).

Acute rheumatism is primarily a disease of school age and often produces serious results. It impairs physical health, causes serious loss of education by preventing attendance at school, is the main cause of organic cardiac disease in early life, and may lead to physical incapacity for years and to death relatively early in adult life. Until it is possible to prevent children becoming the victims of this disease, treatment must be limited to early diagnosis and the use of those measures which are known to hold out the best prospects of limiting the cardiac damage.

These objects can best be effected by the following:—

(1) The establishment of cardio-rheumatic clinics where the diagnosis can be established early and with certainty.

(2) The organization of hospital schools where children can be efficiently treated for so long as may be necessary while education continues.

(3) The compulsory notification of all cases of acute rheumatism, chorea, and rheumatic heart disease.

These steps may ultimately prove desirable for the whole country. At the moment, however, it is suggested that a trial should be made in certain selected centres (e.g. large towns with medical schools) where suitable personnel for staffing clinics and hospital schools would be available. *It must be stressed that these three actions must be taken concurrently and that there would be no object in setting up notification if no clinic were established or in starting a clinic unless it has access to a hospital school.*

The Cardio-Rheumatic Clinic

This should be situated in such a position that it is easy of access from the surrounding district. It should be closely associated with a key hospital, so that good laboratory facilities may be readily available, and the clinical material of the clinic may be utilized for teaching purposes. It should be purely consultative and advisory, with the objects of diagnosis and

supervision, of follow-up, of research, and possibly of the direction of adolescent patients into suitable occupations.

Consultations should be by appointment (where necessary transport should be provided), and school medical officers and general practitioners should be encouraged to attend. The clinic should be held at least once a week, and when needed an evening session should be arranged to meet the requirements of patients in employment. The follow-up and supervision should continue into adult life. This clinic should be staffed by physicians with experience of children's diseases and diseases of the heart. This is essential to enable the clinic to deal with problems arising in the differential diagnosis of rheumatic manifestations and to follow up cardiac cases into adult life. In some centres it may be possible to find one physician who combines these functions, but in others it will be necessary to make use of a team. They should have the help of an assistant physician or registrar. An almoner should be attached to the clinic and adequate secretarial assistance must also be provided. It is desirable that the equipment should include: apparatus for measuring height and weight; an X-ray set for screening and photographing hearts; an electrocardiograph and a technician; a laboratory for sedimentation rates, blood counts, biochemical investigations, etc.

The function of this clinic should be the early diagnosis of acute rheumatism in children and adolescents, and the follow-up of those patients who have had carditis, with the following objects.

(1) Securing the best possible treatment so as to minimize cardiac damage.
(2) Supervising the life and activities of the rheumatic child.
(3) The compilation of reliable data with the object of securing the prevention of acute rheumatism.
(4) The direction of cardiac defectives into suitable occupations.
(5) The education of medical practitioners in the diagnosis of acute rheumatic carditis and cardiac disease generally.
(6) The differentiation of habit spasms and tics from chorea, and " growing pains " from acute rheumatism, which experience has shown form a large part of the work of such a clinic.

Consultations at the homes of children should be made possible. The parents, the family doctor, and the school medical officer should be advised as to fitness for school and any treatment needed. A report should be sent to the family doctor and to the school medical officer.

All cases of acute rheumatism, chorea, and carditis notified should be immediately referred to the clinic unless admitted to the hospital school. School medical officers and general practitioners should be authorized and encouraged to refer to the clinic all children under the age of 16, in whom cardiac murmurs of doubtful significance are found. This is important for two reasons:—

(a) There is often difficulty in deciding whether a given murmur is evidence of past or present carditis or a congenital malformation, or is an " innocent " or " functional " murmur; it is most important that any such doubt should be removed so that unnecessary restrictions need not be imposed.
(b) The proper examination of such cases has a great value from the point of view of research and education.

It is desirable in addition that all children with a history of past rheumatism or chorea, even though they have no signs of cardiac damage, should also be referred to the clinic so that they may be kept under supervision and any relapse noted as soon as possible.

In addition to the report and advice on school and treatment a note should be made as to the necessity for another examination (i.e. in 6 weeks, 3 months, 6 months, etc.) and arrangements made for the child to be summoned to the clinic again at that time.

Hospital Schools

Such hospitals should be established at suitable points throughout the country. They should be hospitals for treatment of children with cardiac rheumatism, but should also be equipped with well-qualified teachers and full school apparatus so that educational facilities may be available for all those inmates who can benefit by education. In addition to continuing general education, each child should be trained as far as may be possible so that he is able to earn a living without the necessity of laborious physical work. By various grades of recreational education both knowledge and manual skill may be acquired.

Where possible these schools should be situated in the country, and they should be large enough to accommodate all cases of acute rheumatism arising in the district served. The stay of each child should be a period of many months. Each hospital school must be supplied with adequate hospital equipment so that nursing of severely ill children can be carried out and so that progress may be recorded—i.e. an electrocardiograph and a technician, an X-ray screen and a suitable laboratory. Each should be a centre for research into ætiological factors and improved treatment of acute rheumatism. In these hospital schools the treatment of acute rheumatism might well be combined with that of other " long stay " children's diseases. This may be particularly necessary in view of the probable shortage of skilled teachers of the necessary type since it is clearly desirable that *all* children suffering from long illnesses should be provided with educational facilities.

Where possible the physician in charge of the cardio-rheumatic clinic should also visit and supervise the hospital school and the assistant physician or registrar should also be attached to the hospital school.

Children will be admitted to the hospital school either direct from their homes immediately on notification, from other hospitals, or from the clinic, and patients should be retained in the hospital school until either (a) their condition is proved not to be due to acute rheumatism or other cause of cardiac damage or (b) it is certain that the infection is quiescent or the condition cured, and the child is fit to live at home. On return they should, whenever possible, attend ordinary schools. Experience has shown that unless there is considerable cardiac enlargement these children are well able to lead a normal school life with full activities. Cases with more severe cardiac damage can attend ordinary schools provided they do no competitive games or drill. P.D. schools of a special type may be of great value by enabling education to continue until the age of 16 and by providing a special vocational training in the final years. But it must be remembered that it is most desirable from the psychological point of view that these children should be brought up as much like normal children as possible. A small number of children may be so severely damaged that it is advisable to transfer them to an institution for chronic invalids if they cannot be adequately cared for at home. Immediately on discharge from the hospital school the child should be referred back to the clinic for supervision.

In view of the striking social incidence of acute rheumatism every effort should be made to promote satisfactory housing for all children and to see that all children receive adequate diet and clothing. Children who have had acute rheumatism should be given special home visits with supervision, and improvement of home environment whenever possible.

The public and the profession should be educated to realize the potential importance of upper respiratory infection however trivial it may appear. Parents of children attending the supervisory clinics should be instructed to report any such infection at once so that proper care may be instituted in an attempt to prevent a rheumatic relapse.

Other Measures

Vocational Training.—Facilities must be made available for vocational training of rheumatic children during their last years at school and on leaving school so that they are trained for

suitable sedentary employment. When this has been done steps must be taken to ensure that when they start work they do in fact obtain such employment.

Notification—The serious results of rheumatic carditis and the importance of early diagnosis and adequate treatment make it imperative that *all cases of suspected acute rheumatism, chorea, and rheumatic heart disease in children under the age of sixteen years* should be made compulsorily notifiable.

It should be the duty of any medical practitioner who suspects that a child or young person is suffering from any of the above-named conditions to notify immediately the local Health Authority.

It should be the duty of the local Health Authority upon the receipt of such notification to refer the child to a cardio-rheumatic clinic, and if necessary to offer to the parents accommodation for the child in a hospital school. It should be possible for the notification to be cancelled if, after examination at the clinic, it is found that the child is not suffering from rheumatism, chorea, or rheumatic heart disease.

Research.—All clinics and hospital schools should be so staffed and equipped that they become active centres of research. This research should be co-ordinated by frequent interchange of ideas amongst the staffs of the various regions. This could perhaps be best facilitated by a co-ordinating committee who should be informed of all research in progress.

SOCIAL AND ECONOMIC CONDITIONS AND THE INCIDENCE OF RHEUMATIC HEART DISEASE

BY

G. H. DANIEL

It has not been the custom of this Journal to review papers of cardiological interest. However this paper, published in the Journal of the Royal Statistical Society (1942) 105, 197, seems to justify making an exception, as it is pertinent to the preceding report and may not have been easily available to all who are interested.

That rheumatic fever and mitral stenosis are more common among the poor than the rich is accepted teaching, supported by much experience and statistical evidence. This, however, fails to show if the difference is due to the effect of income on diet, or on housing, or on other conditions of the environment—material or mental—or might even be explained by hereditary factors. A much more detailed statistical analysis would be needed to isolate these facts.

The Medical Research Council's Report of 1927 sought to evaluate the effects of social and economic conditions by comparing the living conditions of 721 families with children attending hospital for rheumatism with those of 200 families with children attending hospital for non-rheumatic diseases. Of this Daniel says: " It is quite possible that their diseases as well as those of the rheumatic families were related to adverse social and economic circumstances. In that case, comparison could not be expected to reveal the importance of those conditions. The rheumatic families should be compared with the total population."

In 1937 the University of Bristol with the Colston Research Society investigated the circumstances of a random sample (one family in twenty-two) of the Bristol working class population. With the data available the opportunity was taken of comparing the incidence of rheumatism with some of the other social and economic factors; rheumatic heart disease was taken as the significant evidence because this was a more precise criterion.

The number of rheumatic families in each class was divided by the number of families from the sample of the total working-class population in the same class and, to facilitate comparison of incidence between the classes, the result was expressed as a percentage of the proportion between all 341 rheumatic families and 1424 families in the total population sample. This gave for each class the incidence of the disease as a percentage of the average incidence among the entire population studied.

The Bristol data thus yield the following information:

(*a*) Incidence of rheumatic heart disease in each group of working-class families as a percentage of the average incidence for all Bristol working-class families. This will be taken as the dependent variable (Y) and for the sake of brevity it will be referred to as the " incidence."

(*b*) Income available for expenditure on food, clothes, and fuel as a percentage of minimum needs. This independent variable (X1) will be referred to simply as the " net income." It is not, of course, the total family income that is likely to affect susceptibility to disease, as much as the income relative to the needs of the family.

(*c*) Number of rooms per person (X2).

(*d*) Use of a basement room, receipt of meals or milk at school, and membership of a doctor's club.

The conclusions reached about (*b*) and (*c*) are shown in Tables I and II.

TABLE I

INCIDENCE OF RHEUMATIC HEART DISEASE IN RELATION TO NET INCOME

Net income (as a percentage of minimum needs)	Families with rheumatic heart disease	Families in sample of total working-class population	Incidence of rheumatic disease (as a percentage of average incidence	Expected incidence*
Under 100	77	232	139	142
100–120	54	152	148	122
120–140	51	191	112	108
140–160	33	192	72	97
160–180	37	192	81	88
180–200	31	149	87	81
200–220	17	93	76	75
220 and over	41	223	77	70
Total	341	1424	—	—

* Estimated from $Y=4291\ X_1^{-0.737}$

TABLE II

INCIDENCE OF RHEUMATIC HEART DISEASE IN RELATION TO THE NUMBER OF ROOMS PER PERSON

Rooms per person	Families with rheumatic heart disease	Families in sample of total working-class population	Incidence of rheumatic disease (as a percentage of average incidence)	Expected incidence*
Under 0·6	52	130	167	187
0·6–0·8	69	193	149	136
0·8–1·0	53	194	114	107
1·0–1·2	73	335	91	89
1·2–1·4	59	303	81	76
1·4–1·6	10	71	59	66
1·6–1·8	19	138	58	59
1·8 and over	6	60	42	53
Total	341	1424	—	—

* Estimated from $Y=95{\cdot}1\ X_2^{-1.037}$.

The information gathered about the living conditions of the Bristol working-class population allows the following conclusions to be drawn.

(*a*) Differences in incidence of rheumatic heart disease between sections of this population were associated with differences in the family income available after paying for rent, rates, travelling, and other fixed items, expressed as a percentage of minimum needs.

(*b*) Considerable differences in incidence corresponded closely with the variation in the number of rooms used by each family divided by the number of persons in the family.

(*c*) Families which belonged to doctors' clubs suffered less from the disease than did the remainder of the population.

(*d*) There is an indication, though the results are not statistically significant, that families which lived or slept in basement rooms included more cases of the disease than other families.

(*e*) Each one of these relations was true independently of the others.

To what extent these findings based on the Bristol 1937 experience apply to the population of the whole country is unknown. But Bristol is a large town, and there is no apparent reason why the relations found should be confined to it.

This short summary and extract will give some idea of the care with which conclusions have been reached, and may refer some who are interested to the original paper.

MAURICE CAMPBELL

UNUSUAL ELECTROCARDIOGRAM IN DEXTROCARDIA

BY

J. M. HOLFORD

Received February 4, 1944

The case here described was found in the course of a mass radiographic survey.

Case History.—The patient was a well-grown man aged 35 years. He was free from symptoms. He had no significant past medical history. He had always lived a normal life and had been moderately athletic in his school days.

On clinical examination it was found that cardiac pulsation was palpable in the right fourth intercostal space just internal to the mid-clavicular line. Normal heart sounds were heard in this area and between it and the sternum. At the left border of the sternum sounds were faint and further to the left were inaudible. No bruits were heard. There was no dyspnœa, cyanosis, clubbing of the fingers, or other indication of cardiac disorder. Blood pressure was 120/70.

Radiography showed a heart in the position of complete dextrocardia, but otherwise of normal contour. The lung fields were clear. The left dome of the diaphragm was raised. The liver and stomach were not transposed.

Electrocardiographic Findings.—A cardiogram consisting of the three standard limb leads and three chest leads is shown in Fig. 1.

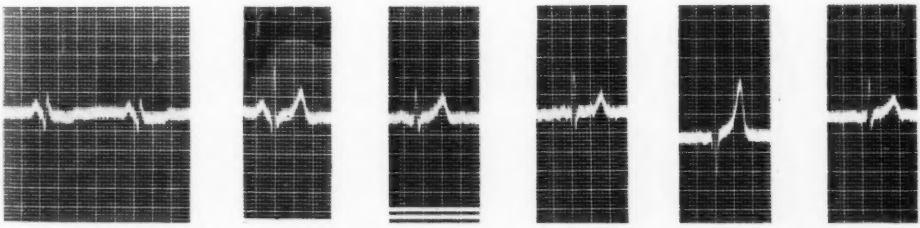

FIG. 1.—The standard leads I, II, and III are followed by CF1, CF2, and CF4 in that order, reading from the left. The chest leads have been taken in the mirror image of the normal position; thus, in CF1, the chest electrode is at the left border of the sternum, in CF2 at the right border, and in CF4 in the right mid-clavicular line; the neutral electrode is on the right leg.

In lead I, P is upright, the QRS complex shows a downward deflection followed by an upward one of approximately equal magnitude, and T is shallow and diphasic. All the remaining leads appear normal.

In their paper on the normal electrocardiogram Chamberlain and Hay (1939) gave no instances of an inverted P wave in lead I, though it was sometimes so small as to be hardly visible. The accepted finding in dextrocardia, with or without situs inversus, is inversion of all waves in lead I. Katz (1941) and Pardee (1942) in their standard text-books of electrocardiography mention no other departure from the normal. Gross (1941), reporting four fresh cases in the course of a comprehensive review of dextrocardia, appears to regard complete inversion in lead I as an invariable accompaniment of the condition. Beaujeu

and Benmussa (1939), writing from Tunis, where dextrocardia is alleged to be much commoner than in Europe, have observed upright P waves in lead I in a case complicated by the tetralogy of Fallot; they specifically state, however, that this does not occur in the absence of congenital heart disease. The low voltage T waves in lead I in the present case are also not mentioned by any of these writers.

I have to thank Surgeon Rear-Admiral F. J. D. Twigg for permission to publish this case.

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AURICULO-VENTRICULAR RHYTHM

BY

R. A. MILLER

Received March 20, 1944

Auriculo-ventricular rhythm is a relatively uncommon and obscure disorder of the heart beat, only diagnosed by means of the electrocardiogram. It occurs when the A-V node assumes control over the ventricles. When this happens, two types of auriculo-ventricular rhythm may arise. One is called A-V nodal rhythm and is recognized by the inverted P waves, which are as a rule either abnormally close to the QRS complex or may follow it. The second type of auriculo-ventricular rhythm is known as A-V dissociation, characterized by the rate of the ventricular contractions being slightly faster than those of the auricles so that there is no constant P–R or R–P interval. Theoretically, the difference between the two rhythms is due to retrograde conduction being present in A-V nodal rhythm in order to allow the auricles as well as the ventricles to respond to the new pace-maker in the A-V node, while in A-V dissociation retrograde conduction from A-V node is blocked so that the auricular contractions must arise above the A-V node, probably in the sino-auricular node (Cutts, 1937; Richardson, 1922). When ventricular bigeminy complicates A-V rhythm it is described as ventricular interference: as reciprocal rhythm when it occurs with A-V nodal rhythm, and as pseudo-reciprocal rhythm when it accompanies A-V dissociation.

To obtain a fuller understanding of these rhythms a record of thirteen such cases will be given and their unusual features will be discussed under the following headings: contributions to ætiology, the mode of onset of A-V rhythm, the significance of associated symptoms, and the value of tests used in differentiating the underlying mechanism of the disorder. Before entering upon any discussion or drawing any conclusions upon the present work, a brief account of the salient features of six of the present series of cases will be related.

Case Histories

Case 1.—Mr. S. C., aged 20, had subacute rheumatic fever and exhibited nodal rhythm for at least four months, 15/10/41 to 5/2/42. The disorder was associated with aortic and mitral disease, and exercise, atropine, and vagal stimulation were all able to produce normal sinus rhythm. The exercise consisted of doing 9512 ft.-lb. work in two minutes. The dose of atropine was 1/30 of a grain given intravenously. The result of the vagal pressure test is recorded in Fig. 1. This shows nodal rhythm,

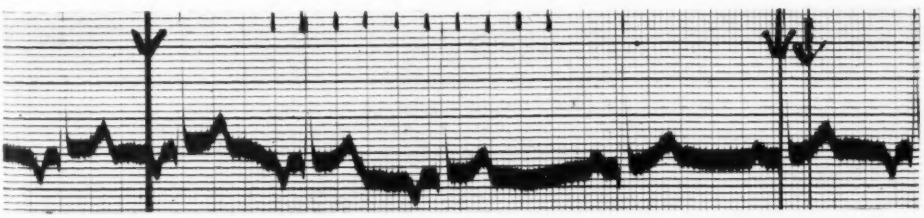

Fig. 1.—*Case* 1. Nodal rhythm changing to normal sinus rhythm by means of carotid sinus pressure, which was maintained between the single thick vertical line and the double thick vertical lines, marked with arrows. The time-marker shows 1/5 and 1/25 sec.

rate 80, followed by normal sinus rhythm when the heart rate had fallen to 50 beats a minute. This change occurred after the application of vagal pressure which is indicated by a thick vertical line. Immediately vagal pressure is removed (denoted by a pair of thick vertical lines) the heart rate rises and the nodal rhythm reappears.

Case 2.—Mrs. M. J., aged 42, came under observation because of faints. Clinically, however, there was no evidence of organic disease, nor did her electrocardiogram (Fig. 2A) signify any cardiac

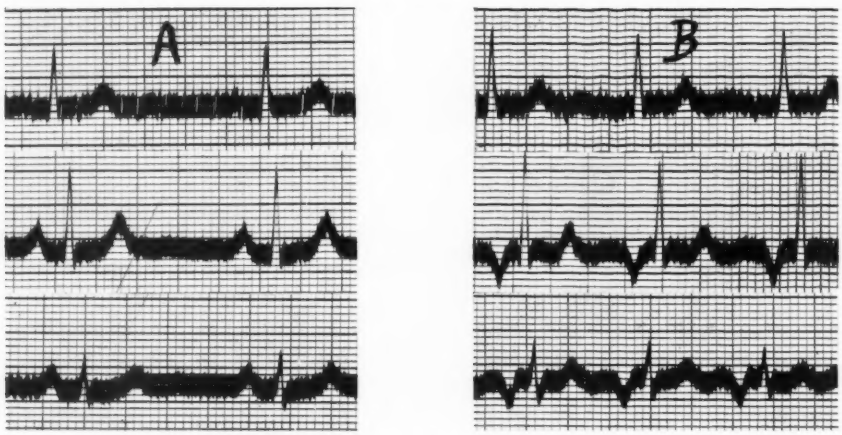

FIG. 2.—*Case* 2. (A) Normal sinus rhythm. (B) A-V rhythm after exercise.

lesion. The same day she did 4694 ft.-lb. work in two minutes and temporarily developed nodal rhythm (Fig. 2B).

Case 3.—Mr. A. F., aged 57, was admitted to hospital on 19/1/40 with essential hypertension and congestive heart failure. At first he had normal sinus rhythm, but later it was converted to A-V dissociation with digitalis therapy. When A-V dissociation was present on 6/2/40 and 16/11/40 the patient was nauseated with digitalis and the drug was withheld while the action of intravenous atropine, 1/50–1/30 grain, was studied on five occasions. In no instance did atropine abolish the abnormal rhythm, but on each occasion the rhythm was complicated by intermittent ventricular bigeminy (Fig. 3A) 62–90 seconds after the injection, and it lasted approximately 22–27 seconds.

FIG. 3.—*Case* 3. (A) A-V dissociation with intermittent ventricular bigeminy due to atropine administration. (B) A-V rhythm with three distinctive ventricular complexes.

During this period the ventricular rate was 43–54 beats a minute It was also noted that there were three ventricular rhythms characterized by their three distinctive QRS complexes and by their inherent rates of 35·8, 41·7 and 37·5 (Fig. 3B), but it was only the last one that was associated with ventricular bigeminy.

Case 4.—Mr. W. B., aged 49, demonstrates how either exercise or atropine abolishes auriculo-ventricular rhythm in an apparently healthy man who probably had the cardiac disorder for 25 years. He had complained of palpitation since he was nine years old and owing to D.A.H. he was discharged

from the Army during the 1914–18 war. At the time of the present observations he still had palpitation and was unduly short of breath on exertion. On examination in 1942 he was a plethoric type of man, but otherwise fit. Electrocardiographically he had nodal rhythm which slowed two or three beats a minute with either right or left carotid sinus pressure. Exercise, amounting to 9861 ft.-lb. work in two minutes raised the heart rate from 65 to 93 and at the same time normal sinus rhythm appeared. Intravenous atropine, 1/30 grain, had the same effect as the exercise test.

Case 5.—Miss M. R., aged 7, was physically fit, but clinically and radiologically there was evidence of a congenital heart with a septal defect. She was proved to have auriculo-ventricular rhythm of long standing, but normal sinus rhythm and sino-auricular block temporarily interrupted it. She was examined on seven occasions when her dominant rhythm was auriculo-ventricular complicated by ventricular bigeminy (Fig. 4A). The latter appeared when the ventricular rate ranged from 42 to 56 provided the Q–P interval was over 0·24 second.

The reaction of the abnormal rhythm to an augmented heart rate was to produce normal sinus

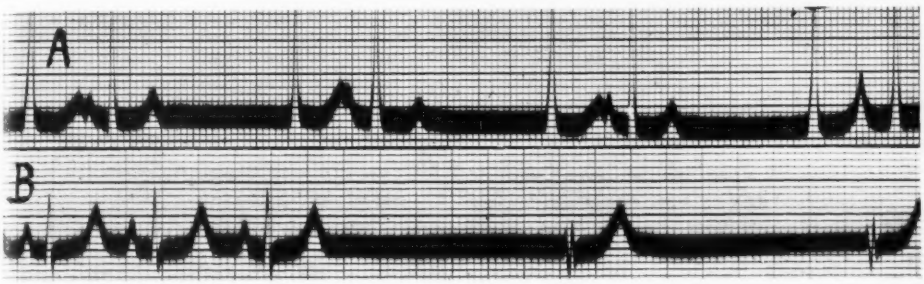

FIG. 4.—*Case* 5. (A) Auriculo-ventricular rhythm with ventricular bigeminy. (B) After physical exercise she developed normal sinus rhythm which was terminated by sino-auricular block and A-V rhythm.

rhythm ; this was done on 6/6/41, after the patient had performed 2188 ft.-lb. work in two minutes : the transformation was again affected on 9/6/41 by injecting atropine sulphate, 1/100 grain, intravenously. It was strange that with the exercise test the maximum rate was not recorded immediately after the work for it rose from 64 to 108 after resting twelve and a half minutes. Yet another phenomenon occurred at this point; the normal sinus rhythm was periodically interrupted by sino-auricular block, which in turn was terminated by an idio-ventricular beat that continued to recur rhythmically for a short period at the rate of 36 to produce nodal rhythm (Fig. 4B).

Vagal stimulation did not alter the auriculo-ventricular rhythm except on one occasion when the auricular rate fell 9 beats a minute.

Case 6.—Mr. K. G., aged 25, was an apparently healthy man who for the past eight weeks had suffered from attacks of faintness and dizziness while blowing his bugle. On examination no physical abnormality was discovered. His electrocardiogram (Fig. 5) revealed normal sinus rhythm

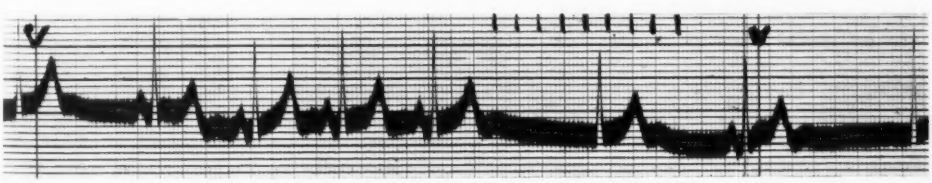

FIG. 5.—*Case* 6. Normal sinus rhythm during inspiration (marked by the thick vertical line and arrow) and A-V dissociation with expiration (marked by the second thick vertical lines and arrow).

with a heart rate of 75 following inspiration (indicated by thick vertical line), but with full expiration A-V dissociation occurred when the heart rate fell to 43 (indicated by the second thick vertical line). When exercise or atropine was given to this patient this abnormality was abolished, although sinus arrhythmia was exceptionally prominent.

ÆTIOLOGY

Both nodal rhythm and auriculo-ventricular dissociation arise through either a depression or destruction of the S-A node or else by enhancing the activity of the A-V node. Other times these factors act collectively. Such changes can be brought about by an alteration in vagal

tone (Bishop, 1921; Cutts, 1937; Gallavardin, 1920; Goodman, 1930; Resnik, 1925; Weiss, 1933), degenerative processes (Cowan, 1939), infective agents (Cutts, 1937; Reid, 1930), and by the administration of atropine, digitalis, and other drugs (Hewlett, 1923; Zeisler, 1932). The rhythm defect, however, may be missed unless exercise is given prior to the electrocardiographic investigation (Case 2).

When ventricular bigeminy complicates the A-V rhythm, the pathology and precipitating factors are similar (Bain, 1939; Bishop, 1921; Blumgart, 1930; Cowan, 1939; Cutts, 1937; Jones, 1927; Katz, 1938; Luten, 1932; and Reid, 1930; also Cases 1, 2, and 5) except that it has not been observed after exercise or with general anæsthesia (Kurtz, 1936). Doubt as to its existence in the dying heart and with asphyxia associated with Cheyne-Stokes breathing also prevails, but, in the former, A-V rhythm may be interrupted by ectopic beats with or without a compensatory pause (Sigler, 1937; Turner, 1931).

When atropine is responsible for A-V rhythm the rate may range from 52 to 100 and it may persist while the heart accelerates (Table I). The figures are obtained by taking electrocardiographic records of nine people before, during and after the intravenous injection of atropine sulphate, 1/30 grain. As intermittent observations were made after the drug had been given, the onset and termination of A-V rhythm was not always accurately known. Nevertheless, it will be seen from Table I that the rate of A-V dissociation at some time of its existence was greater than the initial heart rate. This suggests that the rhythm disorder can occur while the heart rate is augmented. This is demonstrated more conclusively in five cases (3, 6, 7, 8, and 9) where A-V dissociation was recorded while the heart rate was increasing. Such behaviour cannot be said to be due to superimposed organic disease for the disorder sometimes occurred in apparently normal subjects and because normal sinus rhythm always developed with the maximum heart rate.

TABLE I

A-V Dissociation due to Atropine Administration

Case No.	Date	Remarks	Ventricular rate with N.S.R.	Ventricular rate with dissociation
3	4/3/40	Hypertension in congestive failure..	37–60	55.
3	26/11/40	Hypertension in congestive failure..	37–56	52–56
6	25/3/41	Heart normal..	57–95	57–83
7	5/9/40	Heart normal..	79–120	93–96
8	7/6/40	Mitral stenosis	48–57	54–55
8	10/6/40	Mitral stenosis	47–60	54–60
9	27/2/40	Bronchitis and congestive failure ..	79–94	79–94
9	5/3/40	After digitalization	88–120	88–98
10	1/8/40	Heart normal..	60–88	70
11	25/9/41	Aortic incompetence..	62–88	75
12	16/9/40	Angina pectoris	66–94	79
13	8/11/41	Hypertension and heart failure ..	75–115	100

Therefore, in the present series of cases, atropine probably acted more rapidly upon the auriculo-ventricular node than on the sino-auricular node, so as to allow the ventricles to beat faster than the auricles and at the same time to permit both auricle and ventricles to increase their rates. This view on the dual action of atropine is not shared by Wilson (1915) who believed that A-V dissociation due to atropine was due to an initial " inverse " excitation of the vagus which was followed by the later paralytic effect.

Mode of Onset

It is generally believed that the transition from normal sinus rhythm to auriculo-ventricular rhythm takes place without the cognizance of the patient or doctor, for no characteristic signs or symptoms are produced. Nevertheless, suspicion of its development should be aroused

by the onset of a bradycardia. As a rule, the alteration in rhythm is accompanied by a gradual slowing of the heart rate, but in Case 5 there was an abrupt fall in the heart rate from 96 to 36 beats a minute, the change being due to normal sinus rhythm being terminated by S-A block which was followed by an A-V rhythm with a ventricular rate of 36. A similar sequence of events has been described by Weiss and Baker (1933) in their Cases 1 and 2. They achieved the transformation in rhythm by applying carotid sinus pressure to their patients. Therefore, it is probable that alteration in vagal tone explains the change in Case 5 as well as in those mentioned by Weiss and Baker (1933).

The factors that account for ventricular bigeminy being intermittent in A-V rhythm are the rate of the heart and the refractory period of the A-V node and bundle. The latter is determined by measuring the Q–P intervals, or the time which elapses between the initial deflexion of the QRS complex and the beginning of the P wave. The shortest Q–P interval when ventricular bigeminy is present and the longest Q–P interval when the extra beat is absent, are the two important measurements, as it is somewhere between them that the refractory period lies. Details of these investigations are given on Table II and in the history of Cases 3 and 5. They may be summarized by stating firstly that a bradycardia is almost certainly essential for the development of ventricular bigeminy. Secondly, the ventricular rates during which the bigeminy appears will vary from case to case. Thirdly bigeminy is controlled by the refractory period of the A-V node and bundle. It is also clear that an increase in the heart rate is accompanied by a diminution in the refractory period which is analogus to reduction in the P–R interval when the heart rate is augmented in some people with partial heart block.

TABLE II

INFLUENCE OF HEART RATE AND Q–P INTERVAL UPON VENTRICULAR BIGEMINY

Case No.	Date	Ventricular rate per minute	Shortest Q–P interval with bigeminy, seconds	Longest Q–P interval without bigeminy, seconds
Blumgart (Case 2)..	1930	60·0	0·36	0·32
Case 3	13/2/40	55·5	0·32	0·30
Case 3	8/2/40	47·6	0·37	0·35
Case 5	6/6/41	45–48	0·34	0·32
Case 3	6/2/40	44·1	0·40	0·40
Case 3	8/2/40	42·2	0·46	0·46

It is now evident that ventricular bigeminy must have a complex mechanism and it probably arises through the stimulus that activates the auricle passing backwards to re-enter the ventricle to produce an extra ventricular contraction. This sequence of events is known as " re-entry " and it is preferred by most authors to the alternative theory of " parasystole." The latter is based on the supposition that there is a second automatic centre situated low down in the auricle which initiates the extra ventricular contraction (Blumgart, 1930, and Luten, 1932).

SIGNIFICANCE OF SYMPTOMS

It is uncommon for A-V rhythm to produce symptoms; palpitation, however, is sometimes reported (Cowan, 1935; Schotts, 1937; and Thomson, 1941), and it may be associated with ventricular bigeminy (Case 4). At other times faintness and giddiness occur (Katz, 1938, and Case 6) and they are probably due to the accompanying bradycardia that develops suddenly without an adequate compensatory mechanism in the cerebral or the systemic circulation. Such an explanation has been offered for similar symptoms in a person who developed a sudden

bradycardia while normal sinus rhythm was present (Turner, 1931). The prognosis in these cases is the same as for any other symptom experienced during A-V rhythm. That is to say, prognosis is determined entirely by the associated pathological process and not by the abnormal rhythm (Richardson, 1922).

The Value of Tests in Detecting the Defective Mechanism

Three tests have been utilized in an attempt to discover whether or not the A-V conducting tissue is defective. These tests have been developed on the assumption that acceleration of the heart tends to increase metabolic disturbances that may accentuate or reveal abnormal rhythm or conduction, while slowing the heart rate will have the opposite effect. The tests involve the use of atropine, exercise, and vagal stimulation, and are simple to perform. Unfortunately, their value is very limited on account of both vagal dysfunction and organic disease of the cardiac conducting mechanism being sometimes present (Cases 1 and 3). At other times, technical errors, such as failure to repeat a test with greater vigour or with larger doses of atropine in people with persistent A-V rhythm may give rise to fallacies.

The vagal stimulation test is performed by pressure on the carotid sinus. Normally, a heart can react in numerous ways to such a procedure. Moreover, if an alteration in rhythm is produced it cannot always be reproduced (Weiss, 1934). When vagal stimulation is utilized in the investigation of A-V rhythm, there are two responses that are informative; in one A-V rhythm persists throughout the test and is indicative of either vagal dysfunction or a cardiac lesion as the ætiological factor of the rhythm disorder (Blumgart, 1930; Dock, 1928; Luten, 1932; Thomson, 1941; Wedd, 1930; White, 1915; and Cases 4 and 9). In the other response, normal sinus rhythm develops, from which it may be concluded that organic disease of the heart is present. As far as is known, Case 1 is the only example exhibiting these features, but as he had aortic and mitral disease, the test did not aid in the prognosis and treatment of this patient. Therefore, it may be concluded from the above evidence that vagal stimulation is of little or no value in investigating A-V rhythm.

The two tests that are likely to prove of value in A-V rhythm are the exercise test and the atropine test. The former consists of making the patient do so many foot-pounds of work in a given time, while the latter is done by giving atropine, 1/60–1/20 grain, orally or subcutaneously, or intravenously (Bokspan, 1928; Goodman, 1930; Katz, 1938; Luten, 1932; Neslin, 1932; Ritchie, 1923; Wedd, 1941; White, 1915; and Cases 1, 3, 5, and 13). These tests are, however, only warranted or enlightening when the ætiology of A-V rhythm is obscure. In view of this, six apparently healthy people with A-V rhythm (Bishop, 1921; Cutts, 1937; Thomson, 1941; Williams, 1913; and Cases 4 and 6) have been studied, and it has been found that atropine abolished the abnormal rhythm in all but one instance (Williams, 1913). Therefore, the majority exhibited vagal dysfunction, which carries with it a good prognosis. This is even true should the abnormal rhythm be present for eighteen months (Thomson's Case 1, 1941) or possibly twenty years (Case 4). When organic disease is the determining factor in the production of A-V rhythm the expectation of life can not be as good, but it is possible for the rhythm disorder to persist for a year and for health to be maintained over the corresponding period of time (Williams, 1913).

Summary

This paper is based on observations on thirteen cases of auriculo-ventricular rhythm, the salient features of which have been correlated with appropriate facts recorded by others. The outstanding features fall under four headings as follows.

Physical exercise may now be added to the factors that precipitate A-V rhythm. Atropine can produce the rhythm disorder when a tachycardia is present, or when the heart rate is increasing. From these observations it was decided that atropine acted simultaneously and

unequally upon the sino-auricular node and the auriculo-ventricular node in order to produce A-V rhythm.

As a rule, the onset of A-V rhythm cannot be detected by the patient or the physician. Sometimes, however, the patient may complain of giddiness, weakness, or palpitation, and the doctor may note an abrupt fall in the pulse rate. An explanation for these symptoms and signs has been given.

Should A-V rhythm be complicated by ventricular bigeminy, it is generally transitory, but it may be intermittent for months, the onset of bigeminy being partially determined by the length of the Q–P interval and by the site of origin of the ventricular contraction. It should be noted, however, that the critical Q–P interval, below which ventricular bigeminy ceases to appear, varies with the rate of the heart.

The prognosis for people with A-V rhythm is influenced and controlled by systemic disturbances and cardiac disease. The detection of the latter is sometimes possible by utilizing the following tests: vagal pressure, exercise, and atropine, provided such tests are performed and interpreted cautiously. These tests are only warranted when the ætiology of A-V rhythm is obscure. Six such cases have been reviewed. One proved to be due to organic disease and the others to vagal dysfunction. In the former there was no deterioration in health over a period of one year, although the disorder persisted for this period. In those people with abnormal vagal tone as the ætiological factor, it is probably possible to lead an active life for 25 years with intermittent A-V rhythm.

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